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# RESPIRATORY CARE

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## EDITORIALS

To Blow or Not to Blow—That Is the Question

Equality for Women Is Not Fair

## ORIGINAL CONTRIBUTIONS

Performance of a New Screening Spirometer at a Community Health Fair

A Dyspnea Evaluation Protocol for Respiratory Therapists

Intrapulmonary Percussive Ventilation vs Conventional Chest Physiotherapy in Pediatric Atelectasis

Accuracy of Oxygen Analyzers at Subatmospheric Concentrations

Battery Duration of Portable Ventilators

## SPECIAL ARTICLES

COPD Surveillance—United States, 1971-2000

[1]Fink JB, Power J. Am J Respir Crit Care Med 2001;163:A127. [2]Harvey CJ et al. Eur Respir Jl 1997;10:905-909. [3]Fink JB et al. Am J Respir Crit Care Med 1999;159:63-68.
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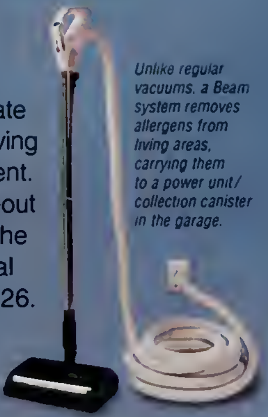

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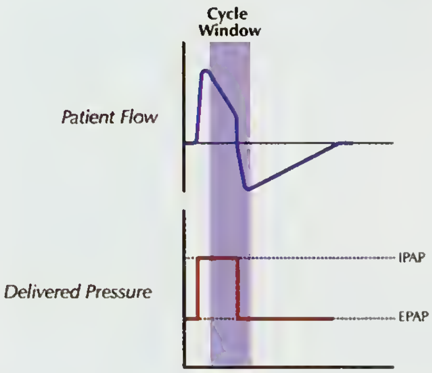

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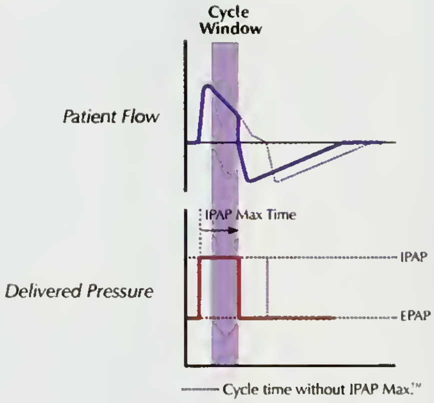

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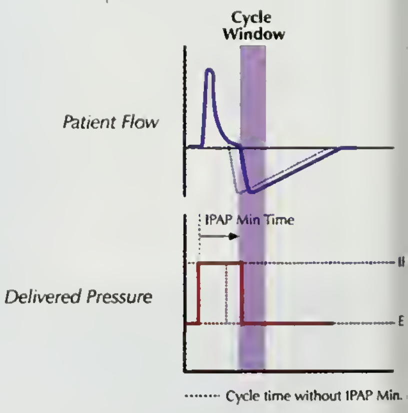

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## Editorials, Commentaries, and Reviews to Note

**Altered Swallowing Physiology and Aspiration in COPD**—Teramoto S, Kume H, Ouchi Y. Chest 2002 Sep;122(3):1104-1105.

**COPD vs. CHF: Use History & Physical Exam Clues to Differentiate & Treat Two Significant Medical Emergencies**—Upchurch J. J Emerg Med Serv JEMS 2002 Sep;27(9):82-94.

**Quality of Life and Functional Parameters in Patients with Chronic Obstructive Pulmonary Disease (COPD): An Update**—Gigliotti F, Grazzini M, Stendardi L, Romagnoli I, Scano G. Respir Med 2002 Jun;96(6):373-374.

**Muscle Mass, Not Body Weight, Predicts Outcome in Patients with Chronic Obstructive Pulmonary Disease**—Mador MJ. Am J Respir Crit Care Med 2002 Sep 15;166(6):787-789.

**Chronic Obstructive Pulmonary Disease**—Kerstjens H, Postma D. Clin Evid 2002 Jun;(7):11344-11357.

**Exercise Carbon Dioxide Retention in Chronic Obstructive Pulmonary Disease: A Case for Ventilation/Perfusion Mismatch Combined with Hyperinflation**—Dempsey JA. Am J Respir Crit Care Med 2002 Sep 1;166(5):634-635.

**Exacerbations of Chronic Obstructive Pulmonary Disease**—Kidney J, McManus T, Coyle PV. Thorax 2002 Sep;57(9):753-754.

**Raising the Profile of Chronic Obstructive Pulmonary Disease with Healthcare Decision-Makers**—Pearson M. Respir Med 2002 Aug;96 Suppl C:S1-S2.

**Bacteria and Exacerbations of Chronic Obstructive Pulmonary Disease** (editorial)—Anthonisen NR. N Engl J Med 2002 Aug 15;347(7):526-527.

**Dyspnea Is a Better Predictor of 5-Year Survival than Airway Obstruction in Patients with COPD**—Nishimura K, Izumi T, Tsukino M, Oga T. Chest 2002 May;121(5):1434-1440.

BACKGROUND: $FEV_1$ is regarded as the most significant correlate of survival in COPD and is used as a measure of disease severity in the staging of COPD. Recently, however, the categorization of patients with COPD on the basis of the level of dyspnea has similarly been reported to be useful in the prediction of health-related quality of life and improvement in exercise performance after pulmonary rehabilitation. Study objectives: We compared the effects of the level of dyspnea and disease severity, as evaluated by airway obstruction, on the 5-year survival rate of patients with COPD. DESIGN AND METHODS: A total of 227 patients with COPD were enrolled in a 5-year, prospective, multicenter study in the Kansai area of Japan, involving 20 divisions of respiratory medicine from various university and city hospitals. RESULTS: After 5 years, 183 patients were available for the follow-up examination (follow-up rate, 81%). The 5-year cumulative survival rate among patients with COPD was 73%. The effect of disease staging, based on the American Thoracic Society (ATS) guideline as evaluated by the percentage of predicted $FEV_1$, on the 5-year survival rate was not significant ($p = 0.08$). However, the level of dyspnea was significantly correlated to the 5-year survival rate ($p < 0.001$). The Cox proportional hazards model revealed that the level of dyspnea had a more significant effect on survival than disease severity based on $FEV_1$. CONCLUSIONS: The categorization of patients with COPD on the basis of the level of dyspnea was more discriminating than staging of disease severity using the ATS guideline with respect to 5-year survival. Dyspnea should be included as one of the variables, in addition to airway obstruction, for evaluating patients with COPD in terms of mortality.

**Mortality After Hospitalization for COPD**—Almagro P, Calbo E, Ochoa de Echaguen A, Barreiro B, Quintana S, Heredia JL, Garau J. Chest 2002 May;121(5):1441-1448.

OBJECTIVES: To identify variables associated with mortality in patients admitted to the hospital for acute exacerbation of COPD. DESIGN: Prospective cohort study. SETTING: Acute-care hospital in Barcelona (Spain). PATIENTS: One hundred thirty-five consecutive patients hospitalized for acute exacerbation of COPD, between October 1996 and May 1997. MEASUREMENTS AND RESULTS: Clinical, spirometric, and gasometric variables were evaluated at the time of inclusion in the study. Socioeconomic characteristics, comorbidity, dyspnea, functional status, depression, and quality of life were analyzed. Mortality at 180 days, 1 year, and 2 years was 13.4%, 22%, and 35.6%, respectively. Sixty-four patients (47.4%) were dead at the end of the study (median follow-up duration, 838 days). Greater mortality was observed in the bivariate analysis among the oldest patients ($p < 0.0001$), women ($p < 0.01$), and unmarried patients ($p < 0.002$). Hospital admission during the previous year ($p < 0.001$), functional dependence (Katz index) [$p < 0.0004$], greater comorbidity (Charlson index) [$p < 0.0006$], depression (Yesavage Scale) [$p < 0.00001$]), quality of life (St. George's Respiratory Questionnaire [SGRQ]) [$p < 0.01$], and $P_{CO_2}$ at discharge ($p < 0.03$) were also among the significant predictors of mortality. In the multivariate analysis, the activity SGRQ subscale ($p < 0.001$; odds ratio [OR], 2.62; confidence

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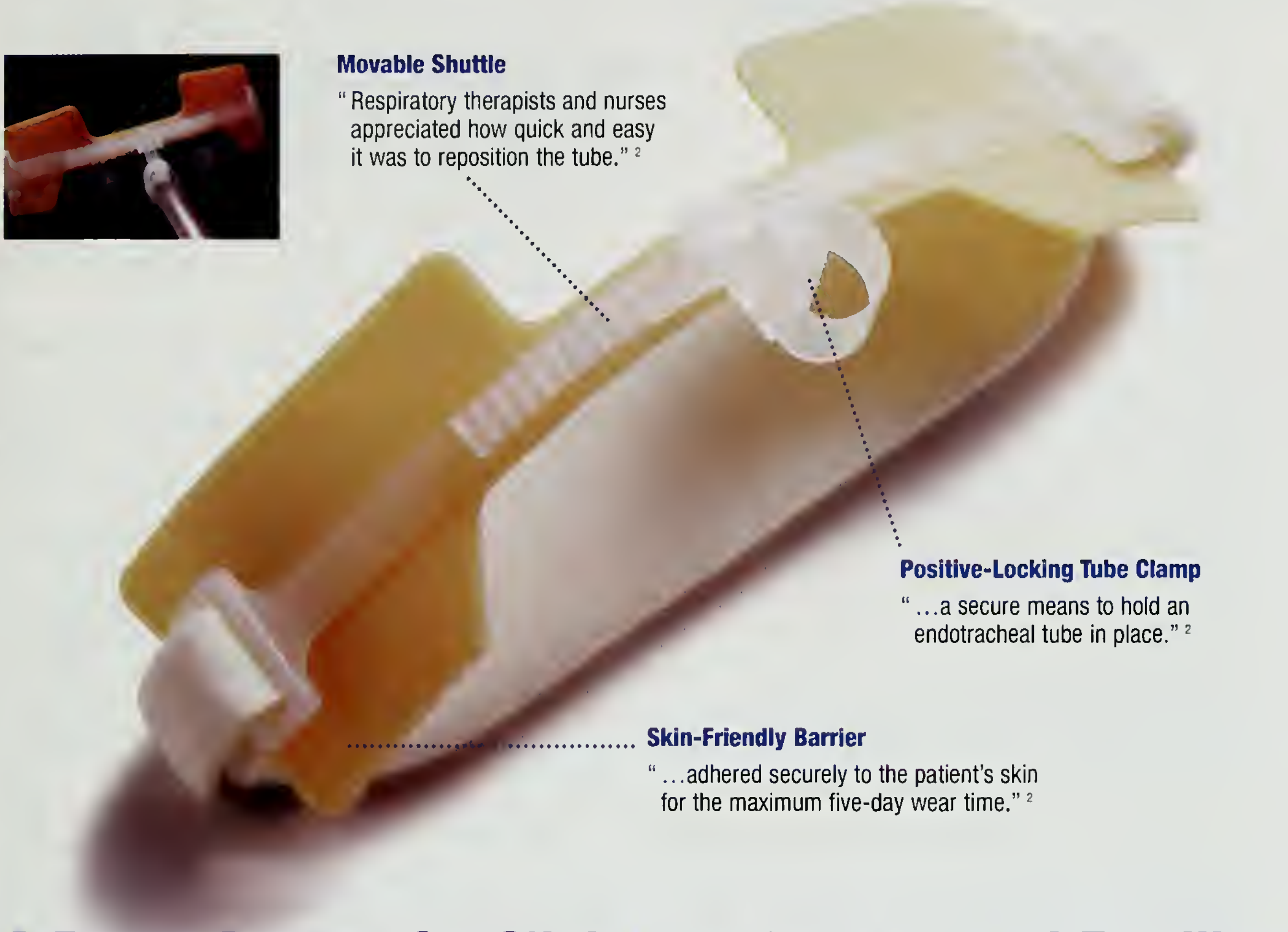

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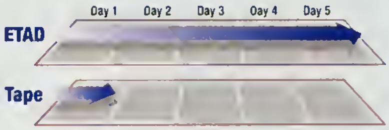

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1. Clinical evaluation of an Oral Endotracheal Tube Attachment Device (ETAD) Hollister, 1995.
2. Cunneen, Jane A different approach to endotracheal tube stabilization. Hollister, 1996

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interval [CI], 1.43 to 4.78), comorbidity ($p < 0.005$; OR, 2.2; CI, 1.26 to 3.84), depression ($p < 0.004$; OR, 3.6; CI, 1.5 to 8.65), hospital readmission ($p < 0.03$; OR, 1.85; CI, 1.26 to 3.84), and marital status ($p < 0.0002$; OR, 3.12; CI, 1.73 to 5.63) were independent predictors of mortality. CONCLUSIONS: Quality of life, marital status, depressive symptoms, comorbidity, and prior hospital admission provide relevant information of prognosis in this group of COPD patients.

**Pharmacoeconomic Evaluation of Acute Exacerbations of Chronic Bronchitis and COPD**—Miravitlles M, Murio C, Guerrero T, Gisbert R. Chest 2002 May;121(5):1449-1455.

BACKGROUND: Although exacerbations are the main cause of medical visits and hospitalizations of patients with chronic bronchitis and COPD, little information is available on the costs of their management. OBJECTIVE: This study attempted to determine the total direct costs derived from the management of exacerbations of chronic bronchitis and COPD in an ambulatory setting. METHOD: A total of 2,414 patients with exacerbated chronic bronchitis and COPD were recruited from 268 general practices located throughout Spain. Patients were followed up for 1 month. RESULTS: A total of 507 patients (21%) relapsed; of these, 161 patients (31.7%) required attention in emergency departments and 84 patients (16.5%) were admitted to the hospital. The total direct mean cost of all exacerbations was $159; patients who were hospitalized generated 58% of the total cost. Cost per failure was $477.50, and failures were responsible for an added mean cost of $100.30/exacerbation. Exacerbations of the 1,130 patients with COPD had a mean cost of $141. Sensitivity analysis showed that a 50% reduction in the failure rate (from 21 to 10.5%) would result in a total cost of exacerbation of $107 (33% reduction). CONCLUSION: Exacerbations of chronic bronchitis and COPD are costly, but the greatest part of costs derives from therapeutic failures, particularly those that end in hospitalization.

**COPD in Perspective**—Petty TL. Chest 2002 May;121(5 Suppl):116S-120S.

In the last 200 years or so, the recognition, diagnosis, and understanding of the pathogenesis of COPD have evolved considerably. Over the past few decades, various definitions of COPD and its "components" also have developed. Despite this, however, the treatment options for patients with this relentlessly progressive disorder are relatively limited. In the mid-19th century, the introduction of the spirometer yielded a powerful tool for the diagnosis of COPD. The currently available small, cheap spirometers hold great promise to help patients and their physicians closely monitor lung function. Early recognition of the close associations among emphysema and, more recently, small airways disease, and impaired airflow is discussed. This review also stresses the importance of the identification of COPD in its initial stages and the early onset of appropriate treatment. The therapy for COPD has changed in the last 40 years. Drug therapies in the 1960s included potassium iodide and ephedrine. Corticosteroids were not used, and oxygen therapy and exercise were actually contraindicated. Modern therapy for COPD is now more systematic and includes the use of bronchodilators and corticosteroids to improve airflow, in addition to oxygen therapy, pulmonary rehabilitation and, in selected patients, lung volume reduction surgery. The causal link between the chronic inhalation of tobacco smoke and COPD is beyond doubt, and smoking cessation remains the most important goal for patients. It is hoped that new, more effective therapies will soon be available for the treatment of this disabling disorder to provide improvement in symptoms and patient quality of life and to reduce or stop the rate of disease progression and mortality in patients with COPD.

**COPD: Epidemiology, Prevalence, Morbidity and Mortality, and Disease Heterogeneity**—Mannino DM. Chest 2002 May;121(5 Suppl):121S-126S.

COPD continues to cause a heavy health and economic burden both in the United States and around the world. Some of the risk factors for COPD are well-known and include smoking, occupational exposures, air pollution, airway hyperresponsiveness, asthma, and certain genetic variations, although many questions, such as why < 20% of smokers develop significant airway obstruction, remain. Precise definitions of COPD vary and are frequently dependent on an accurate diagnosis of the problem by a physician. These differences in the definition of COPD can have large effects on the estimates of COPD in the population. Furthermore, evidence that COPD represents several different disease processes with potentially different interventions continues to emerge. In most of the world, COPD prevalence and mortality are still increasing and likely will continue to rise in response to increases in smoking, particularly by women and adolescents. Resources aimed at smoking cessation and prevention, COPD education and early detection, and better treatment will be of the most benefit in our continuing efforts against this important cause of morbidity and mortality.

**Systemic Effects in COPD**—Wouters EF, Creutzberg EC, Schols AM. Chest 2002 May;121(5 Suppl):127S-130S.

The pathogenesis and clinical manifestations of COPD are not restricted to pulmonary inflammation and structural remodeling. Rather, this disorder is associated with clinically significant systemic alterations in biochemistry and organ function. The systemic aspects of COPD include oxidative stress and altered circulating levels of inflammatory mediators and acute-phase proteins. Indeed, an impaired endogenous oxidant-antioxidant balance has been reported in patients experiencing exacerbations of COPD, and others have observed altered circulating levels of several cytokines and adhesion molecules in patients with stable disease. As in other chronic inflammatory conditions, weight loss, muscle wasting, and tissue depletion are commonly seen in COPD patients. Selective wasting of fat-free mass coupled with impaired respiratory and peripheral muscle function and a reduced capacity for exercise occur in COPD patients. Indeed, weight loss may directly impact poor prognosis in COPD patients. The mechanisms underlying weight loss and muscle wasting are incompletely understood but likely involve an imbalance in ongoing processes of protein degradation and replacement. This may include alterations in the relative levels or activities of endocrine hormones such as insulin, growth hormone, testosterone, and glucocorticoids. Furthermore, chronic systemic inflammation involving cytokines such as interleukin-1 and tumor necrosis factor-alpha may be associated with these hormonal changes and muscle wasting in COPD patients. This review includes a discussion of the mechanisms of skeletal muscle fiber protein metabolism/catabolism, the potential roles of endogenous cytokines in protein loss, and the possibility that novel drugs that inhibit cytokine signaling may provide benefits by reducing muscle wasting and cachexia, thereby improving the prognosis and quality of life among COPD patients.

**Exacerbations: Etiology and Pathophysiologic Mechanisms**—Wedzicha JA. Chest 2002 May;121(5 Suppl):136S-141S.

Some patients with COPD are prone to frequent exacerbations, which are an important determinant of health status. Such patients have elevated airway cytokine levels, suggesting the presence of increased inflammation that may increase their susceptibility to exacerbation. The inflammatory response during a COPD exacerbation is variable, but increases in interleukin-6 levels during the exacerbation are related to the presence of a common cold. Rhinovirus infection is the most important etiologic factor in COPD exacerbations and is an important target for preventive therapy. The reduction of COPD exacerbations will have an important impact on the considerable morbidity and mortality associated with COPD.

**John Hutchinson's Mysterious Machine Revisited**—Petty TL. Chest 2002 May;121(5 Suppl):219S-223S

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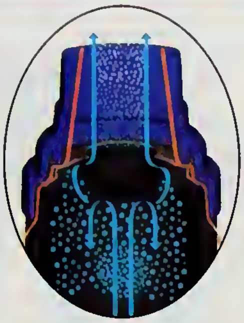

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invented the spirometer to measure what he called the vital capacity, ie, the capacity to live. Much later, the concept of the timed vital capacity, which became known as the $FEV_1$, was added. Together, these two numbers, vital capacity and $FEV_1$, are useful in identifying patients at risk of many diseases, including COPD, lung cancer, heart attack, stroke, and all-cause mortality. This article cites some of the rich history of the development of spirometry, and explores some of the barriers to the widespread application of simple spirometry in the offices of primary care physicians.

**Respiratory Infections with *Pseudomonas Aeruginosa* in Children with Cystic Fibrosis: Early Detection by Serology and Assessment of Risk Factors**—West SE, Zeng L, Lee BL, Kosorok MR, Laxova A, Rock MJ, et al. JAMA 2002 Jun 12;287(22):2958-2967.

CONTEXT: Patients with cystic fibrosis (CF) are susceptible to lower respiratory tract infections with *Pseudomonas aeruginosa* and typically acquire this organism in early childhood. Once *P aeruginosa* infection is established, eradication may be impossible, and progressive lung disease often aggravates morbidity and mortality risks. The ability to diagnose CF by genetic testing at birth makes it possible to determine the temporal sequence of events that result in *P aeruginosa*-associated pulmonary infections. OBJECTIVE: To evaluate the longitudinal relationship between the production of an antibody response against *P aeruginosa* and clinical factors associated with *P aeruginosa* pulmonary infections in patients with CF diagnosed in early life. DESIGN, SETTING, AND PATIENTS: Serum samples and oropharyngeal cultures (protocol cultures) were obtained at 6-month intervals from April 15, 1985, to April 15, 2000 (or for up to 180 months depending on their enrollment date) from 68 patients at 2 centers in Madison and Milwaukee, Wis, diagnosed through the Wisconsin CF Neonatal Screening Project, a longitudinal cohort study. Additional cultures were obtained at examining physicians' discretion (all cultures). MAIN OUTCOME MEASURES: Time to serum IgG, IgA, and IgM antibody titer of at least 1:256 against *P aeruginosa*, assessed by enzyme-linked immunosorbent assay using cell lysate, exotoxin A, and elastase as antigens; time to organism isolation from respiratory samples; time to Wisconsin Cystic Fibrosis Radiograph (WCXR) score of 5 or more. RESULTS: The median time to an antibody titer of at least 1:256 was 17.8, 24.2, and 70.9 months for cell lysate, exotoxin A, and elastase, respectively. The rise of anti-cell lysate and anti-exotoxin A titers to 1:256 or more occurred a mean of 11.9 ($p<0.001$) and 5.6 ($p=0.04$) months, respectively, before the isolation of *P aeruginosa* for all cultures and 18.2 ($p<0.001$) and 11.9 ($p=0.006$) months, respectively, before protocol cultures. There was no significant difference between the rise of anti-cell lysate and anti-exotoxin A titer and a WCXR score of 5 or more ($p=0.24$ and.32, respectively). Treatment with long-term, non-*Pseudomonas* oral antibiotics and integration of CF infants with older, chronically infected patients were associated with a significantly increased risk of *P aeruginosa* pulmonary infection. CONCLUSIONS: In CF patients diagnosed through neonatal screening, *P aeruginosa* pulmonary infections occurred 6 to 12 months before the organism was isolated from respiratory secretions. The longitudinal monitoring of *P aeruginosa* antibody titers, in concert with WCXR score, should facilitate diagnosis and treatment of *P aeruginosa* pulmonary infections in young children with CF.

**Using Tobacco-Industry Marketing Research to Design More Effective Tobacco-Control Campaigns**—Ling PM, Glantz SA. JAMA 2002 Jun 12;287(22):2983-2989.

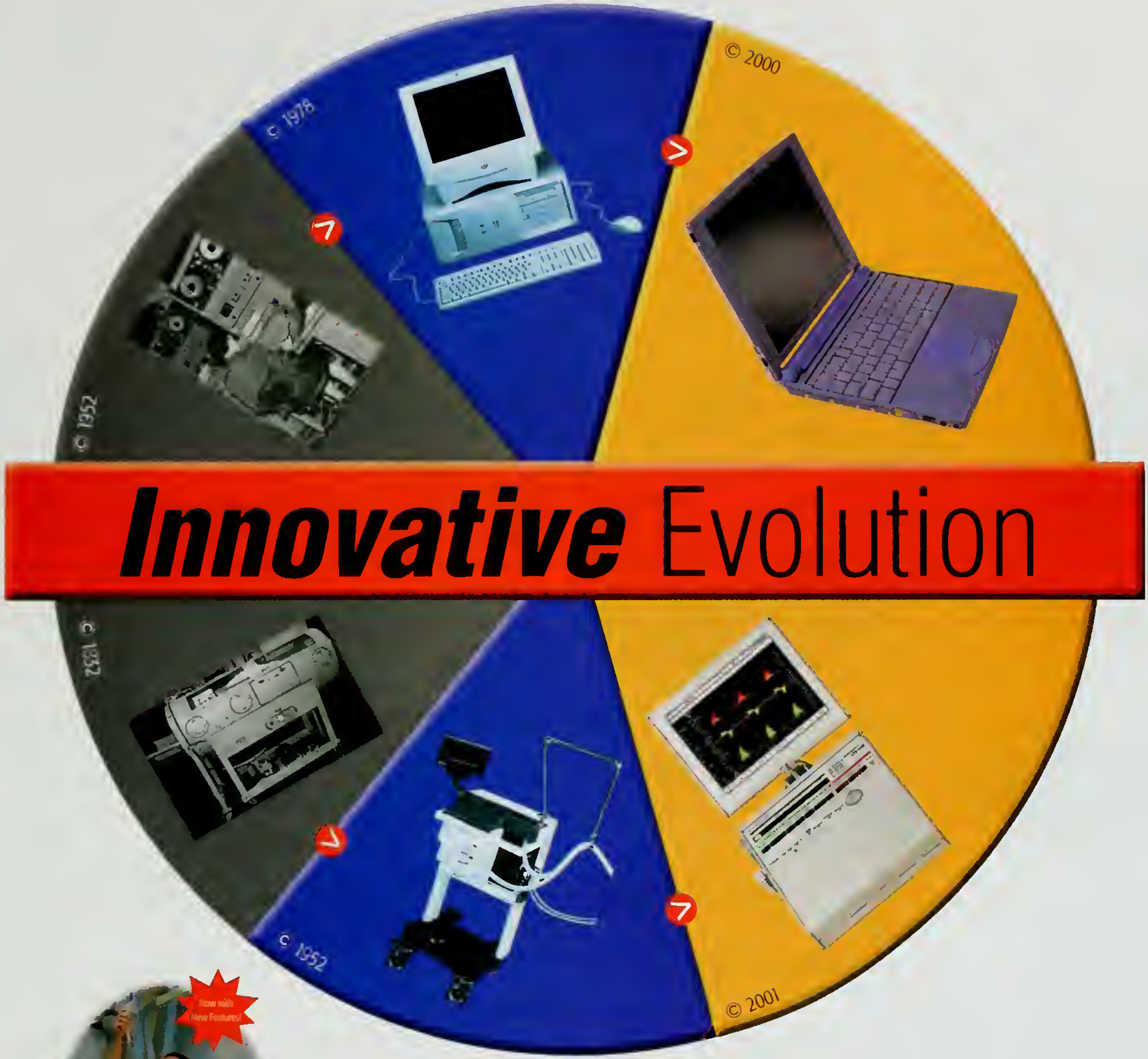
To improve tobacco-control efforts by applying tobacco-industry marketing research and strategies to clinical and public health smoking interventions, we analyzed previously secret tobacco-industry marketing documents. In contrast to public health, the tobacco industry divides markets and defines targets according to consumer attitudes, aspirations, activities, and lifestyles. Tobacco marketing targets smokers of all ages; young adults are particularly important. During the 1980s, cost affected increasing numbers of young and older smokers. During the 1990s, eroding social acceptability of smoking emerged as a major threat, largely from increasing awareness of the dangers of secondhand smoke among nonsmokers and smokers. Physicians and public health professionals should use tobacco-industry psychographic approaches to design more relevant tobacco-control interventions. Efforts to counter tobacco marketing campaigns should include people of all ages, particularly young adults, rather than concentrating on teens and young children. Many young smokers are cost sensitive. Tobacco-control messages emphasizing the dangers of secondhand smoke to smokers and nonsmokers undermine the social acceptability of smoking.

**Efficacy of a Nicotine Lozenge for Smoking Cessation**—Shiffman S, Dresler CM, Hajek P, Gilburt SJ, Targett DA, Strahs KR. Arch Intern Med 2002 Jun 10;162(11):1267-1276.

BACKGROUND: Since nicotine gum was introduced in the 1980s, nicotine replacement therapy has become the most widely used pharmacological smoking cessation treatment. Some smokers prefer acute oral forms, but many smokers reject chewing gum. We tested the safety and efficacy of a new nicotine polacrilex lozenge for smoking cessation. METHODS: Double-blind, placebo-controlled, randomized clinical trial with parallel arms testing 2- and 4-mg nicotine lozenges. Smokers ($n = 1818$) were assigned to a lozenge dose on the basis of nicotine dependence, assessed by time to the first cigarette of the day. Low-dependence smokers were randomized to receive the 2-mg nicotine ($n = 459$) or placebo ($n = 458$) lozenge; high-dependence smokers, the 4-mg nicotine ($n = 450$) or placebo ($n = 451$) lozenge. We assessed abstinence at 6, 12, 24, and 52 weeks and analyzed craving and withdrawal symptoms. RESULTS: Treatment with the nicotine lozenge resulted in significantly greater 28-day abstinence at 6 weeks, for the 2-mg (46.0% vs. 29.7%; odds ratio [OR], 2.10; 95% confidence interval [CI], 1.59-2.79; $p<0.001$) and the 4-mg (48.7% vs. 20.8%; OR, 3.69; 95% CI, 2.74-4.96; $p<0.001$) lozenges, compared with placebo. Significant treatment effects were maintained for a full year. Smokers who used more lozenges achieved significantly better treatment effects. Use of the active lozenge also resulted in reduced craving and withdrawal. Most adverse events were moderate and resembled those seen with nicotine gum. CONCLUSION: The nicotine lozenge is a safe and effective new treatment for smoking cessation in low- and high-dependence smokers.

**Independent Validation of the Sleep Apnoea Quality of Life Index**—Lacasse Y, Godbout C, Series F. Thorax 2002 Jun;57(6):483-488.

BACKGROUND: Obstructive sleep apnoea (OSA) affects important domains of quality of life which remain unexplored by conventional sleep recordings. The objective of this study was to examine the measurement properties (both discriminative and evaluative) of the Sleep Apnoea Quality of Life Index (SAQLI), a new OSA specific quality of life questionnaire. METHODS: Consecutive patients recently diagnosed with OSA completed a French version of the SAQLI twice over a 3 month period. Its construct validity and responsiveness were tested by comparing baseline and change scores obtained in each domain (symptoms, activities, emotions, social interactions) with those of questionnaires measuring related constructs (SF-36, Epworth Scale, Beck Depression Inventory, and Symptom Checklist 90). The symptoms scores were also correlated with physiological measures obtained at baseline polysomnographic recording. RESULTS: Forty seven patients (40 men) of mean (SD) age 53 (10) years and mean (SD) apnoea/hypopnoea index 38 (21) participated in the study. During the study period 33 of the 47 patients were treated for OSA (31 with nasal CPAP, one with uvulopalatopharyngoplasty, and one with an oral appliance). Moderate to high correlations were found between the scores in each domain of the SAQLI and the cor-



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responding instruments. There were significant differences in change scores between patients who were treated and those who were not, moderate correlations between SAQLI change scores and changes in the corresponding instruments, and no correlation between the symptoms scores and the baseline nocturnal features. Most of these correlations met the a priori predictions made regarding their magnitude. CONCLUSION: The SAQLI has strong construct validity and is responsive to change in quality of life but has the disadvantage of having to be administered by an interviewer.

**Effect of CPAP on Intrinsic PEEP, Inspiratory Effort, and Lung Volume in Severe Stable COPD**—O'Donoghue FJ, Catcheside PG, Jordan AS, Bersten AD, McEvoy RD. Thorax 2002 Jun;57(6):533-539.

BACKGROUND: Intrinsic positive end expiratory pressure (PEEPi) constitutes an inspiratory threshold load on the respiratory muscles, increasing work of breathing. The role of continuous positive airway pressure (CPAP) in alleviating PEEPi in patients with severe stable chronic obstructive pulmonary disease is uncertain. This study examined the effect of CPAP on the inspiratory threshold load, muscle effort, and lung volume in this patient group. METHODS: Nine patients were studied at baseline and with CPAP increasing in increments of 1 cm $H_2O$ to a maximum of 10 cm $H_2O$. Breathing pattern and minute ventilation (I), dynamic PEEPi, expiratory muscle activity, diaphragmatic (PTPdi/min) and oesophageal (PTPoes/min) pressure-time product per minute, integrated diaphragmatic (EMGdi) and intercostal EMG (EMGic) and end expiratory lung volume (EELV) were measured. RESULTS: Expiratory muscle activity was present at baseline in one subject. In the remaining eight, PEEPi was reduced from a mean (SE) of 2.9 (0.6) cm $H_2O$ to 0.9 (0.1) cm $H_2O$ ($p<0.05$). In two subjects expiratory muscle activity contributed to PEEPi at higher pressures. There were no changes in respiratory pattern but I increased from 9.2 (0.6) L/min to 10.7 (1.1) L/min ($p<0.05$). EMGdi remained stable while EMGic increased significantly. PTPoes/min decreased, although this did not reach statistical significance. PTPdi/min decreased significantly from 242.1 (32.1) cm $H_2O$.s/min to 112.9 (21.7) cm $H_2O$.s/min). EELV increased by 1.1 (0.3) L ($p<0.01$). CONCLUSION: High levels of CPAP reduce PEEPi and indices of muscle effort in patients with severe stable COPD, but only at the expense of substantial increases in lung volume.

**The Pulmonary Physician in Critical Care # 6: The Pathogenesis of ALI/ARDS**—Bellingan GJ. Thorax 2002 Jun;57(6):540-546.

An understanding of the pathogenesis of ARDS is essential for choosing management strategies and developing new treatments. The key mediators involved in the inflammatory and fibroproliferative responses are reviewed and the mechanisms which regulate these responses are highlighted.

**Central Sleep Apnoea Syndrome in Patients with Chronic Heart Disease: A Critical Review of the Current Literature**—Kohnlein T, Welte T, Tan LB, Elliott MW. Thorax 2002 Jun;57(6):547-554.

The prevalence, prognosis, clinical presentation, pathophysiology, diagnosis, and treatment of the central sleep apnoea syndrome (CSAS) are reviewed and its relationship with congestive heart failure (CHF) is discussed. Adequately powered trials are needed with survival and health status as end points to establish whether correction of sleep related breathing abnormalities improves the outcome in patients with CHF.

**Sleep Disordered Breathing and Pregnancy**—Edwards N, Middleton PG, Blyton DM, Sullivan CE. Thorax 2002 Jun;57(6):555-558.

Many changes in the respiratory system occur during pregnancy, particularly during the third trimester, which can alter respiratory function during sleep, increasing the incidence and severity of sleep disordered breathing. These changes include increased ventilatory drive and metabolic rate, reduced functional residual capacity and residual volume, increased alveolar-arterial oxygen gradients, and changes in upper airway patency. The clinical importance of these changes is indicated by the increased incidence of snoring during pregnancy, which is likely also to reflect an increased incidence of obstructive sleep apnoea/hypopnoea syndrome. For the respiratory physician asked to review a pregnant patient, the possibility of sleep disordered breathing should always be considered. This review first examines the normal physiological changes of pregnancy and their relationship to sleep disordered breathing, and then summarises the current knowledge of sleep disordered breathing in pregnancy.

**Specific Airway Resistance in 3-Year-Old Children: A Prospective Cohort Study**—Lowe L, Murray CS, Custovic A, Simpson BM, Kissen PM, Woodcock A; NAC Manchester Asthma and Allergy Study Group. Lancet 2002 Jun 1;359(9321):1904-1908.

BACKGROUND: The development of a method to assess lung function in young children may provide new insight into asthma development. Plethysmographic measurement of specific airway resistance ($sR_{aw}$) is feasible in this age group. We aimed to identify risk factors associated with low lung function in early childhood in a prospective birth cohort. METHODS: Children were prenatally assigned to risk group according to parental atopic status (high risk, both parents atopic; medium risk, one parent atopic; low risk, neither parent atopic) and followed prospectively until age 3 years. We measured $sR_{aw}$ in 503 symptom-free children using whole-body plethysmography during tidal breathing. FINDINGS: 803 of 868 children attended the clinic, of whom 503 obtained satisfactory sR(aw) readings. 200 who wheezed at least once during first 3 years of life had significantly higher $sR_{aw}$ than the 303 who had never wheezed (mean difference 5.8%, 95% CI 2.2-9.3, p=0.002). For children who had never wheezed there were significant differences in $sR_{aw}$ between risk groups ($p<0.001$). Children at high risk (n=87) had a higher $sR_{aw}$ (geometric mean 1.17 kPa/s, 1.12-1.22) than children at medium risk (n=162; 1.02 kPa/s, 1.00-1.05) and at low risk (54; 1.04 kPa/s, 0.99-1.11). Atopic children (n=62) had significantly higher $sR_{aw}$ (1.15 kPa/s, 1.09-1.21) than those who were not atopic (232; 1.05 kPa/s, 1.02-1.07, p=0.002). For non-atopic children, those at high risk (58) had higher $sR_{aw}$ (1.13kPa/s, 1.07-1.18) than those at medium risk (125, 1.01kPa/s, 0.98-1.05) or at low risk (49, 1.04 kPa/s, 0.97-1.10, p=0.003). We showed a significant interaction between history of maternal asthma and child's atopic status (p=0.006). INTERPRETATION: Even in the absence of respiratory symptoms, children of atopic parents and those with personal atopy have impaired lung function in early life.

**pH in Expired Breath Condensate of Patients with Inflammatory Airway Diseases**—Kostikas K, Papatheodorou G, Ganas K, Psathakis K, Panagou P, Loukides S. Am J Respir Crit Care Med 2002 May 15;165(10):1364-1370.

Endogenous airway acidification, as assessed by pH in expired breath condensate, has been implicated in asthma pathophysiology. We measured pH in breath condensate of patients with inflammatory airway diseases in stable condition and examined its relationship with the inflammatory process (as assessed by differential cell counts in induced sputum), oxidative stress (as assessed by $H_2O_2$ and 8-isoprostane), and nitric oxide metabolism (as assessed by total nitrate/nitrite). We studied 40 patients with bronchial asthma (20 with moderate disease, forced expiratory volume in 1 second 60 [10]% SD predicted), 20 patients with bronchiectasis, 20 patients with chronic obstructive pulmonary disease (COPD), and 10 normal subjects. Mean (95% confidence intervals) pH values were significantly lower in patients with COPD and bronchiectasis compared with patients with asthma and control subjects (7.16, 7.09-7.23

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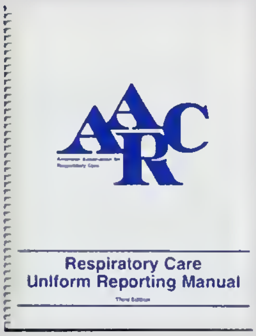

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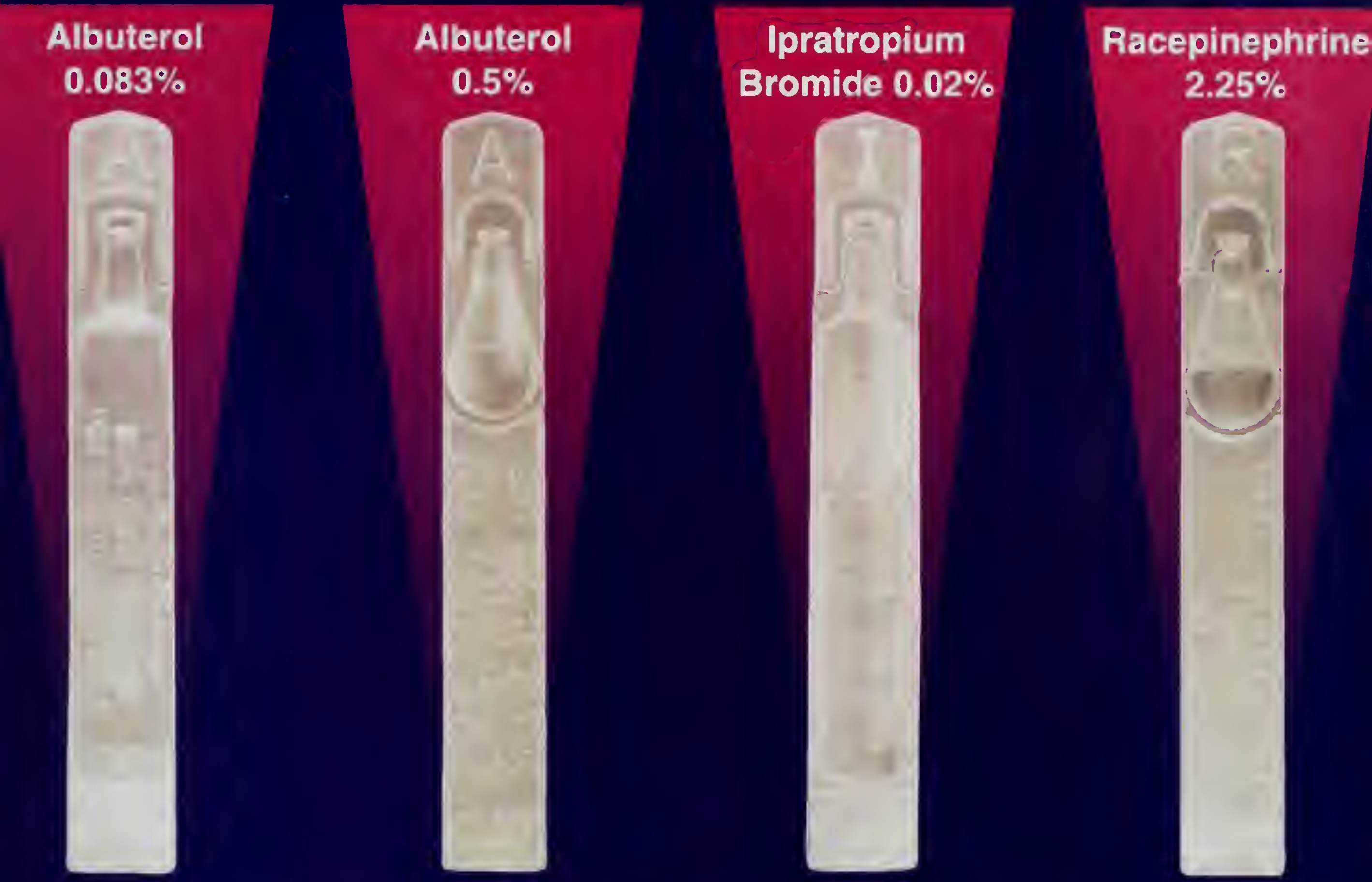
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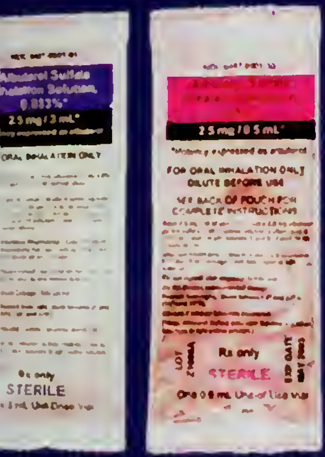

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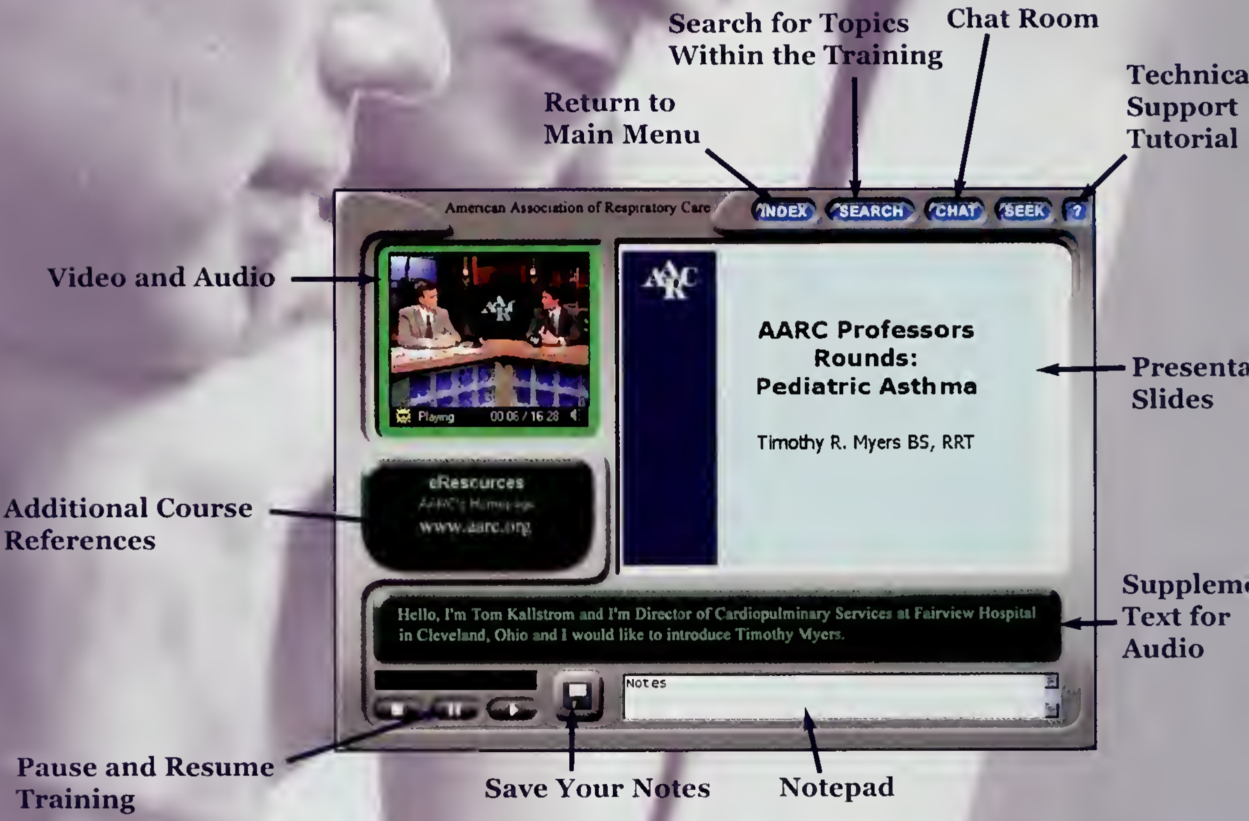
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and 7.11, 7.04-7.19 versus 7.43, 7.35-7.52 and 7.57, 7.51-7.64, respectively, $p < 0.0001$). Patients with moderate asthma had significantly lower values compared with mild and control subjects. In patients with COPD and bronchiectasis, the values of pH were significantly correlated with both sputum neutrophilia and oxidative stress. Respectively, in patients with moderate asthma, a significant correlation was observed between pH and sputum eosinophilia, total nitrate/nitrite, and oxidative stress. The pH of the expired breath condensate might be a simple, noninvasive, inexpensive, and easily repeatable procedure for the evaluation of the inflammatory process in airway diseases.

**Systemic Effect Comparisons of Six Inhaled Corticosteroid Preparations**—Martin RJ, Szefler SJ, Chinchilli VM, Kraft M, Dolovich M, Boushey HA, et al. Am J Respir Crit Care Med 2002 May 15;165(10):1377-1383.

The goal of this study was to establish a reliable method to evaluate systemic bioavailability and to determine equisystemic effects (microgram dose producing equal systemic cortisol suppression) of inhaled corticosteroids (ICS). Steroid naive asthma subjects ($n = 156$) were enrolled at six centers. A 1-week doubling dose design was used for each of six ICS and matched placebos for a total of four doses. Systemic effect was evaluated by hourly plasma cortisol concentrations (8 P.M. to 8 A.M.), 12- and 24-hour urine cortisol concentrations, and a morning blood osteocalcin. The area under the concentration-time curve for hourly cortisol concentrations was the best outcome variable to assess systemic effect. For the six ICS and matching placebos (beclomethasone-chlorofluorocarbon [CFC], budesonide dry powder inhaler [DPI], fluticasone DPI, fluticasone-CFC metered dose inhaler [MDI], flunisolide-CFC, and triamcinolone-CFC), only the placebo group and fluticasone DPI did not demonstrate a significant dose-response effect. Thus microgram comparison of all ICS could only be performed at a 10% cortisol suppression: flunisolide-CFC - 936; triamcinolone-CFC - 787; beclomethasone-CFC - 548; fluticasone DPI - 445; budesonide DPI - 268; fluticasone-CFC MDI - 111. This study represents the first step in evaluation of ICS efficacy based on equisystemic (cortisol suppression) effects of a given ICS, rather than doses judged arbitrarily to be comparable on a microgram basis.

**Pre/Postbronchodilator Interrupter Resistance Values in Healthy Young Children**—Beydon N, Amsallem F, Bellet M, Boule M, Chaussain M, Denjean A, et al. Am J Respir Crit Care Med 2002 May 15;165(10):1388-1394.

The interrupter technique estimates flow resistance. It entails occlusion of the airways during tidal breathing while flow and mouth pressure are recorded. This noninvasive technique is easy to use in young children. The aim of the present study was to measure inspiratory and expiratory interrupter resistance ($Rint_{insp}$, $Rint_{exp}$) before and after bronchodilator administration in young healthy white children. We designed a multicenter study using a standardized procedure for Rint measurements. Centers in five French cities studied 91 children (48 boys and 43 girls; height, 92 to 129 cm; mean age 5.3 ± 1.4 years). Mean values were not significantly different for $Rint_{insp}$ and $Rint_{exp}$ ($0.78 \pm 0.21$ versus $0.78 \pm 0.20$ Kpa • $L^{-1}$ • second). However, the difference between $Rint_{insp}$ and $Rint_{exp}$ decreased significantly with age and being positive before 5 years and negative later on ($p < 0.02$). $Rint_{insp}$ and $Rint_{exp}$ decreased significantly with height ($Rint_{insp}$ [Kpa • $L^{-1}$ • second] = 2.289 - 1.37. $10^{-2}$ • H [cm], $Rint_{exp}$ [Kpa • $L^{-1}$ • second] = 2.021 - 1.12.$10^{-2}$ • H [cm]; $p < 0.001$). Bronchodilator (salbutamol) administration significantly decreased $Rint_{insp}$ and $Rint_{exp}$ ($p < 0.001$). Bronchodilator-induced changes (% of predicted values) in mean $Rint_{insp}$ and mean $Rint_{exp}$ were -15% (95% confidence interval, -46 to +15%) and -12% (95% confidence interval, -46 to +22%), respectively. Sex did not affect pre- or postbronchodilator values. Data from the present study may prove useful for testing lung function in young children with respiratory disorders who failed to cooperate with forced expiratory maneuvers.

**Cardiorespiratory Effects of Inelastic Chest Wall Restriction**—Miller JD, Beck KC, Joyner MJ, Brice AG, Johnson BD. J Appl Physiol 2002 Jun;92(6):2419-2428.

We examined the effects of chest wall restriction (CWR) on cardiorespiratory function at rest and during exercise in healthy subjects in an attempt to approximate the cardiorespiratory interactions observed in clinical conditions that result in restrictive lung and/or chest wall changes and a reduced intrathoracic space. Canvas straps were applied around the thorax and abdomen so that vital capacity was reduced by >35%. Data were acquired at rest and during cycle ergometry at 25 and 45% of peak workloads. CWR elicited significant increases in the flow-resistive work performed on the lung (160%) and the gastric pressure-time integral (>400%) at the higher workload, but it resulted in a decrease in the elastic work performed on the lung (56%) compared with control conditions. With CWR, heart rate increased and stroke volume (SV) fell, resulting in >10% fall in cardiac output at rest and during exercise at matched workloads ($p < 0.05$). Blood pressure and catecholamines were significantly elevated during CWR exercise conditions ($p < 0.05$). We conclude that CWR significantly impairs SV during exercise and that a compensatory increase in heart rate does not prevent a significant reduction in cardiac output. $O_2$ consumption appears to be maintained via increased extraction and a redistribution of blood flow via sympathetic activation.

**Altered Diaphragm Contractile Properties with Controlled Mechanical Ventilation**—Sassoon CS, Caiozzo VJ, Manka A, Sieck GC. J Appl Physiol 2002 Jun;92(6):2585-2595.

This study shows that, over time, diaphragm inactivity with controlled mechanical ventilation (CMV) decreases diaphragm force and produces myofibril damage contributing to the reduced force. We measured in vivo and in vitro diaphragm contractile and morphological properties in 30 sedated rabbits grouped ($n = 6$) as follows: 1 or 3 days of CMV, 1 or 3 days of 0 $cmH_2O$ continuous positive airway pressure, and control. The CMV rate was set sufficient to suppress diaphragm electrical activity. Compared with the control group, phrenic-stimulated maximum transdiaphragmatic pressure did not decrease with continuous positive airway pressure but decreased to 63% after 1 day of CMV and to 49% after 3 days of CMV. The in vitro tetanic force decreased to 86% after 1 day of CMV and to 44% after 3 days of CMV. After 3 days of CMV, significant myofibril damage occurred in the diaphragm but not in the soleus. The decrease in tetanic force correlated with the volume density of abnormal myofibrils. We conclude that CMV had a detrimental effect on diaphragm contractile properties.

**Mast-Cell Infiltration of Airway Smooth Muscle in Asthma**—Brightling CE, Bradding P, Symon FA, Holgate ST, Wardlaw AJ, Pavord ID. N Engl J Med 2002 May 30;346(22):1699-1705.

BACKGROUND: Asthma and eosinophilic bronchitis are characterized by similar inflammatory infiltrates in the submucosa of the lower airway. However, eosinophilic bronchitis differs from asthma in that there is no variable airflow obstruction or airway hyperresponsiveness in the former condition. We tested the hypothesis that there were differences between the two conditions in the microlocalization of mast cells within the airway smooth muscle. METHODS: Immunohistochemical analysis of bronchial-biopsy specimens was completed in 17 subjects with asthma, 13 subjects with eosinophilic bronchitis, and 11 normal controls recruited from two centers. RESULTS: Both groups with disease had a similar degree of submucosal eosinophilia and thickening of the basement membrane and lamina reticularis. By contrast, the number of tryptase-positive mast cells in the bundles of airway smooth muscle from subjects with asthma (median, 5.1 mast cells per square millimeter of smooth muscle [range, 0 to 33.3]) was substantially higher than that in subjects with eosinophilic bronchitis (median, 0 mast cells per square millimeter;

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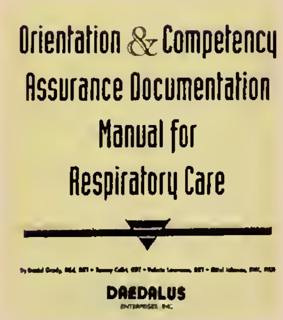
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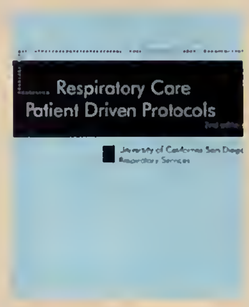

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range, 0 to 4.8) and that in normal controls (median, 0 mast cells per square millimeter [range, 0 to 6.4]; p<0.001 for the comparison among the three groups). T cells and eosinophils were not usually seen in the airway smooth muscle in any of the groups. CONCLUSIONS: The infiltration of airway smooth muscle by mast cells is associated with the disordered airway function found in asthma.

**Nurse-Staffing Levels and the Quality of Care in Hospitals**—Needleman J, Buerhaus P, Mattke S, Stewart M, Zelevinsky K. N Engl J Med 2002 May 30;346(22):1715-1722.

BACKGROUND: It is uncertain whether lower levels of staffing by nurses at hospitals are associated with an increased risk that patients will have complications or die. METHODS: We used administrative data from 1997 for 799 hospitals in 11 states (covering 5,075,969 discharges of medical patients and 1,104,659 discharges of surgical patients) to examine the relation between the amount of care provided by nurses at the hospital and patients' outcomes. We conducted regression analyses in which we controlled for patients' risk of adverse outcomes, differences in the nursing care needed for each hospital's patients, and other variables. RESULTS: The mean number of hours of nursing care per patient-day was 11.4, of which 7.8 hours were provided by registered nurses, 1.2 hours by licensed practical nurses, and 2.4 hours by nurses' aides. Among medical patients, a higher proportion of hours of care per day provided by registered nurses and a greater absolute number of hours of care per day provided by registered nurses were associated with a shorter length of stay (p=0.01 and p<0.001, respectively) and lower rates of both urinary tract infections (p<0.001 and p=0.003, respectively) and upper gastrointestinal bleeding (p=0.03 and p=0.007, respectively). A higher proportion of hours of care provided by registered nurses was also associated with lower rates of pneumonia (p=0.001), shock or cardiac arrest (p=0.007), and "failure to rescue," which was defined as death from pneumonia, shock or cardiac arrest, upper gastrointestinal bleeding, sepsis, or deep venous thrombosis (p=0.05). Among surgical patients, a higher proportion of care provided by registered nurses was associated with lower rates of urinary tract infections (p=0.04), and a greater number of hours of care per day provided by registered nurses was associated with lower rates of "failure to rescue" (p=0.008). We found no associations between increased levels of staffing by registered nurses and the rate of in-hospital death or between increased staffing by licensed practical nurses or nurses' aides and the rate of adverse outcomes. CONCLUSIONS: A higher proportion of hours of nursing care provided by registered nurses and a greater number of hours of care by registered nurses per day are associated with better care for hospitalized patients.

**Is More Neonatal Intensive Care Always Better? Insights from a Cross-National Comparison of Reproductive Care**—Thompson LA, Goodman DC, Little GA. Pediatrics 2002 Jun;109(6):1036-1043.

BACKGROUND: Despite high per capita health care expenditure, the United States has crude infant survival rates that are lower than similarly developed nations. Although differences in vital recording and socioeconomic risk have been studied, a systematic, cross-national comparison of perinatal health care systems is lacking. OBJECTIVE: To characterize systems of reproductive care for the United States, Australia, Canada, and the United Kingdom, including a detailed analysis of neonatal intensive care and mortality. Design/Methods. Comparison of selected indicators of reproductive care and mortality from 1993-2000 through a systematic review of journal and government publications and structured interviews of leaders in perinatal and neonatal care. RESULTS: Compared with the other 3 countries, the United States has more neonatal intensive care resources yet provides proportionately less support for preconception and prenatal care. Unlike the United States, the other countries provided free family planning services and prenatal and perinatal physician care, and the United Kingdom and Australia paid for all contraception. The United States has high neonatal intensive care capacity, with 6.1 neonatologists per 10 000 live births; Australia, 3.7; Canada, 3.3; and the United Kingdom, 2.7. For intensive care beds, the United States has 3.3 per 10 000 live births; Australia and Canada, 2.6; and the United Kingdom, 0.67. Greater neonatal intensive care resources were not consistently associated with lower birth weight-specific mortality. The relative risk (United States as reference) of neonatal mortality for infants <1000 g was 0.84 for Australia, 1.12 for Canada, and 0.99 for the United Kingdom; for 1000 to 2499 g infants, the relative risk was 0.97 for Australia, 1.26 for Canada, and 0.95 for the United Kingdom. As reported elsewhere, low birth weight rates were notably higher in the United States, partially explaining the high crude mortality rates. CONCLUSIONS: The United States has significantly greater neonatal intensive care resources per capita, compared with 3 other developed countries, without having consistently better birth weight-specific mortality. Despite low birth weight rates that exceed other countries, the United States has proportionately more providers per low birth weight infant, but offers less extensive preconception and prenatal services. This study questions the effectiveness of the current distribution of US reproductive care resources and its emphasis on neonatal intensive care.

**Spontaneous Pneumothorax During Pregnancy**—Gorospe I, Puente S, Madrid C, Novo S, Gil-Alonso JL, Guntinas A. South Med J 2002 May;95(5):555-558.

Spontaneous pneumothorax complicating pregnancy is rare. Only 41 cases have been previously published. We describe a case of spontaneous pneumothorax successfully treated with tube thoracostomy during the 38th week of pregnancy. Under epidural anesthesia, the patient had vaginal delivery of a healthy male infant 36 hours after tube thoracostomy.

**Ventilator-Associated Pneumonia in Pediatric Intensive Care Unit Patients: Risk Factors and Outcomes**—Elward AM, Warren DK, Fraser VJ. Pediatrics 2002 May;109(5):758-764.

OBJECTIVES: To determine the rates, risk factors, and outcomes of ventilator-associated pneumonia in pediatric intensive care unit (PICU) patients. METHODS: A prospective cohort study was conducted at the St Louis Children's Hospital PICU on all patients who were admitted to the PICU from September 1, 1999, to May 31, 2000, except those who died within 24 hours, were ≥18 years of age, or were neonatal intensive care unit patients on extracorporeal membrane oxygenation. The primary outcome measured was the development of ventilator-associated pneumonia. Secondary outcomes were death and hospital and PICU length of stay. Multiple logistic regression analysis was performed to determine independent predictors for ventilator-associated pneumonia. RESULTS: There were 34 episodes of ventilator-associated pneumonia in 30 patients of 911 admissions (3.3%) and 595 (5.1%) mechanically ventilated patients. The mean ventilator-associated pneumonia rate was 11.6/1000 ventilator days. By logistic regression analysis, genetic syndrome (odds ratio [OR]: 2.37; 95% confidence interval [CI]: 1.01-5.46), reintubation (OR: 2.71; 95% CI: 1.18-6.21), and transport out of the PICU (OR: 8.90; 95% CI: 3.82-20.74) independently predicted ventilator-associated pneumonia. CONCLUSIONS: Ventilator-associated pneumonia occurs at significant rates among mechanically ventilated PICU patients and is associated with processes of care. Additional studies are necessary to develop interventions to prevent ventilator-associated pneumonia.

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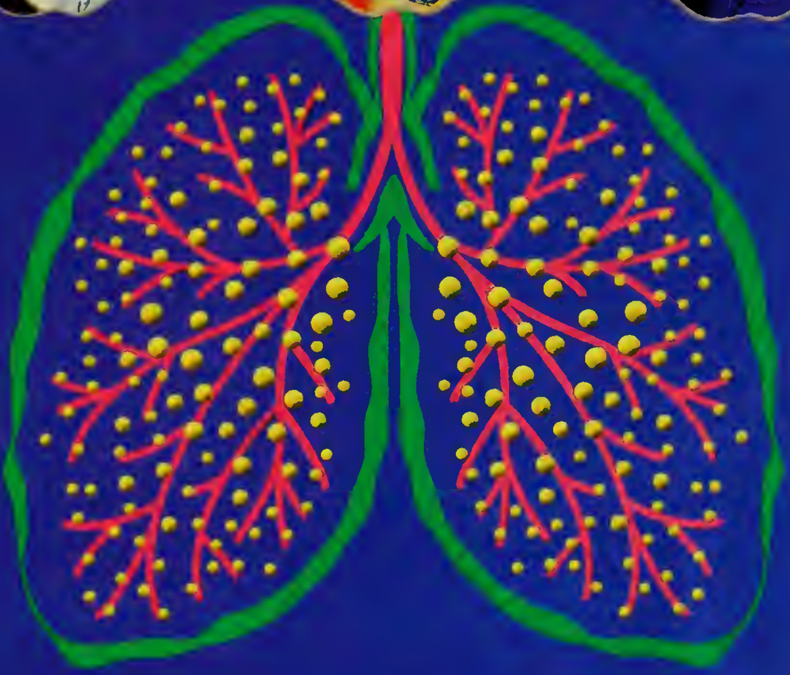

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[1] Wildhaber JH, [illegible]. Schweiz Med Wochenschr [illegible]; 128:1223-1228

[2] Nelson Laboratories, Holding Chamber Comparative and Characterization Study

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# Editorials

## To Blow or Not to Blow—That Is the Question

To be, or not to be—that is the question—

Shakespeare, *Hamlet*, III, i, 57

Our man Hamlet was clearly in a quandary when he uttered those words, but a similar quandary faces us today when we come to the question of whether we should screen for lung disease by performing spirometry.

Long before the formation of the National Lung Health Education Program, members of the pulmonary community had frequently called for the wider use of spirometry to stem the tide of rising mortality due to lung disease.[1] Yet it would appear that those calls for the general use of spirometry have largely fallen on deaf ears. Why?

SEE THE ORIGINAL STUDY ON PAGE 1150

Current standards of medical practice hold that if one suspects heart disease or if a patient is considered at risk for heart disease, then obtaining an electrocardiogram or, at the least, measuring blood pressure is indicated. No physician would even consider not measuring blood pressure, even with a patient who is seen only for a routine visit. So why is it that spirometry is not a routine test? Indeed, it is unclear why the spirometer has not joined the thermometer, sphygmomanometer, chest radiograph, and electrocardiograph as a prominent tool in the physician's office. Perhaps we can gain some insight into this paradox if I relate to you a recent experience.

As part of a steering committee for an upcoming conference, I attended a meeting to discuss the election of speakers on the latest approaches to the management of chronic obstructive pulmonary disease. One of the committee members is a practicing physician in the area, who was participating via conference call from his office. When the inclusion of spirometry as a conference topic came up, we asked him if that topic would be attractive to the private physician and also did he routinely use spirometry in his office. He immediately went on the defensive and stated that he was too busy; he had 3 examination rooms full of patients, no time to do spirometry, and, "Besides, if you can't do it right, why do spirometry at all!" I thought to ask him if he felt the same way about blood pressure, height, and weight measurements, but held my tongue. Why did our busy pulmonary physician not routinely use spirometry in his practice, when we now know the merits of this clinical test? I believe there are 4 major misconceptions or perceived obstacles that keep physicians and other health care workers from using this exceptionally useful test.

### 1. *Spirometry Is a Poor Test and of Little Benefit*

Nothing could be further from the truth. If you perform spirometry or teach about it, I strongly encourage you to read the National Lung Health Education Program's recent position paper.[2] The take-home message is that spirometry is one of the best clinical tests we have. For example, how many clinical tests do you know of that reproduce with a coefficient of variation of ± 2–3%?[3,4] Did you know that spirometry (forced vital capacity [FVC]) beat out blood pressure as a predictor of heart disease in the Framingham Study?[5] And did you know that spirometry (forced expiratory volume in the first second [$FEV_1$]) is the best predictor of mortality due to all causes?[6,7] Clearly, spirometry is not only a *good* clinical test, it might just be an *outstanding* clinical test.

### 2. *The Equipment Is Bulky, Not Very Good, and Expensive*

This used to be true, but not any longer. The efforts of the American Thoracic Society to improve spirometers and spirometry[3] have all but eliminated poor spirometer performance. That is not to say that any given spirometer is always working correctly; spirometers are more complex than sphygmomanometers, making them more prone to problems. However, most problems are solved by a quality control program of device calibration and testing of laboratory personnel.[2,8] The bulkiness of spirometers has disappeared with the advent of flow sensors, lap-top computers, and microprocessor chips. Now a perfectly good spirometer can be the size of a hand-held video game. The cost has fallen also. In this issue of RESPIRATORY CARE the report by Schoh et al[9] used such a modern spirometer, which cost about $500, with 394 people at a health fair, and with great success. The bulkiness, accuracy, and cost barriers to spirometry are gone.

### 3. *Spirometry Is Hard to Do Right*

If spirometry is that hard to do right, then why are so many scientific reports written and published in which the major outcome variable is spirometry values? If spirometry is so hard to do right, why did Schoh et al[9] choose to measure $FEV_1$ at a health fair to test 394 people? A pulmonary function laboratory that I once directed was accused of slowing down patient flow by our insistence on performing spirometry prior to the patient seeing the physician—apparently a novel concept! We set up a quality-assurance/quality-control project that showed that a spirometry study that included bronchodilation took about 15 minutes. Spirometry alone (but with time taken for height, weight, and data entry) took a mere 5–7 minutes. Most physicians typically see 8–12 patients in a half-day clinic. Given that you can charge for the test. . . . . . well, you get the picture.

The real Achilles heel of spirometry is that, unlike other routine clinical tests, it requires active and maximal participation by the subject, and one of the keys to successful spirometry is the person administering it. Administering spirometry takes good coaching skills that not everyone has.[3,7] Remember, though, that even substandard spirometry tests are still interpretable,[3,4] and failure to perform spirometry properly is one indicator of disease.[10] Indeed, the report by Schoh et al[9] demonstrates that it is very feasible to conduct spirometry in the general public in a suboptimal setting. So it is hard to believe that spirometry is so time-consuming or hard to do right that this should be a real barrier to the use of spirometry.

### 4. *What Do All the Numbers Mean and What Do I Do with Them?*

I submit that this is the real barrier to the general use of spirometry. I am often asked for references about spirometry interpretation, and, sad to say, there are limited offerings. While there are many articles on theory, equipment, and performance, there is little to be found in textbooks on fundamental interpretation of test results. This state of affairs is best observed when the new first-year pulmonary fellows arrive each July. When presented with a flow-volume loop, or even just $FEV_1/FVC$, they are at a total loss as to how to interpret these findings This is in sharp contrast to when they are presented with a 12-lead electrocardiogram, which they handle as though they were born cardiologists. So why the difference? The answer is simple. It's the education they receive. Most tell me that they receive little if any instruction in lung function testing, or its interpretation, in medical school or during residency, whereas from the beginning they are exposed to advanced measures of heart performance and their interpretation.

Currently, mortality due to lung disease is ranked number 4 and is projected to rise to number 3 by 2020.[11,12] This dramatic rise in death rates due to lung disease comes in face of the fact that the other 6–8 leading causes of mortality in this country continue to fall. And while the number of patients with doctor-diagnosed chronic obstructive pulmonary disease is estimated at 10 million, the number of people in the general population with abnormal lung function is estimated at 24 million; accordingly, the underdiagnosis of lung disease may approach some 14 million persons.[12]

The best way to meet these challenges is education. Yet our doctors, respiratory therapists, and other health-related professionals are not receiving even cursory education in the use and value of spirometry, in spite of the fact that spirometry is one of the best clinical tests we have. This failure is the fault of our education system. The pulmonary community has not made it a priority to educate others in the value, performance, and, most of all, the interpretation of even basic office spirometry. And the situation was the same more than 20 years ago, when it was observed:

> Perhaps even greater responsibility for the near absence of the use of pulmonary function in the prevention of chronic limitation of airflow must be borne by the expert in pulmonary medicine, especially in his relation to the nonspecialist. If evaluation of pulmonary function is to play a significant role in the prevention of chronic limitation of airflow, it is the pulmonary specialist who is in the best position to educate the rest of the health professions; yet the pulmonary specialist is no more likely to use pulmonary function for prevention than the nonpulmonary specialist.[1]

Or perhaps, as the comic strip character Pogo would say, "We have met the enemy and they is us." So what are *we* going to do about it?

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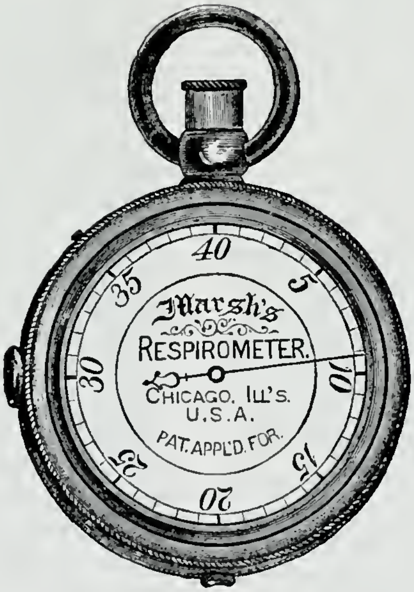

From *The American Armamentarium Chirurgicum*
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# Equality for Women Is Not Fair

"You've come a long way, baby," the theme of many cigarette ads directed toward women, emphasizing the glamour and sexiness of smoking, has now paid off. A recent study by the Centers for Disease Control (CDC),[1] which is reprinted in this issue of RESPIRATORY CARE, showed that chronic obstructive pulmonary disease (COPD) death among women more than equaled that among men in the year 2000 (59,936 vs 59,118). This is more than equality—it's a disaster!

A number of studies have suggested that smoking women are more susceptible to developing COPD than are men.[2–4] Women who are susceptible may develop COPD at an earlier age and with less duration or intensity of smoking, and women have a greater degree of nonspecific bronchial hyperreactivity than men, as demonstrated in the Lung Health Study.[5] So women should be very alert to the risk of developing COPD and, for that matter, related lung cancer.

SEE THE SPECIAL ARTICLE ON PAGE 1184

It appears that the tobacco industry may be stalking women. Their emphasis on women in recent advertising campaigns is an example. Remember the long brown cigarette ads? "How can you smoke a long brown cigarette?" the man asks. "Easy," replies the woman, "I want more of a good thing." Another scene shows a woman with a long cigarette. "That cigarette's so long, we'll miss the wedding," the bridesmaid says. "I'm the bride; they'll wait." A favorite in my series of slides I sometimes present at grand rounds as "Seduced by Smoking" shows a man and a woman under an umbrella in a rainstorm. "How do you keep such a long cigarette dry?" he asks. "I only date men with big umbrellas," she replies. Give me a break!

Though only 1 in 5 smokers develops COPD, no one knows who will develop the disease, unless there is a strong family history or occupational risk. It is well known that COPD has few or even no symptoms until it becomes far advanced. The alarming rise in COPD deaths among women is only half the problem. COPD deaths among men also continue to rise. If mortality trends continue, there will be more than 120,000 COPD deaths in 2002. COPD is now the fourth most common cause of death and the only disease in the top 10 killers that continues to rise. This is despite advances in care of advanced COPD, including oxygen in selected cases, pulmonary rehabilitation, and improved methods of managing acute respiratory failure.[6]

The recently released CDC study[1] indicates that as many as 50% of patients with COPD are undiagnosed today. A similar conclusion was drawn from the National Health and Nutrition Examination Survey study.[7] Thus, the major challenge is obvious: to identify mild-to-moderate stages of lung disease, as a basis for intervention.

The National Lung Health Education Program (NLHEP) was launched in 1997 as a major grassroots initiative designed to identify and treat patients with mild-to-moderate stages of disease.[8] *"Test Your Lungs, Know Your Numbers"* is the motto of the NLHEP. The NLHEP is in partnership with the American Association for Respiratory Care. Thus, some 130,000 respiratory therapists are the foot soldiers for the NLHEP, working in nearly every hospital in the United States. Today a grassroots effort for early identification of COPD and related disorders is underway. NLHEP recommends spirometric testing of all current or former smokers age 45 or older and of anyone with chronic cough, dyspnea on exertion, mucus hypersecretion, or wheeze.[9] The Lung Health Study demonstrated that both men and women who successfully stop smoking have an initial improvement in air flow, as measured by forced expiratory volume in the first second, followed by a slow decline over 5 years, compared with those who continued to smoke, who have a much more rapid rate of decline.[10] The spirometer is the key instrument for diagnosis. It must be used in all primary care physicians' offices and in the offices of many specialists who see patients with dyspnea, such as cardiologists.

Although the tobacco industry has been giving preferential treatment to women to entice them to start smoking, this is not fair, because women are so susceptible to the harmful effects of tobacco. Today some progress is being made in modifying youth smoking behavior and there has been a slight decline in student smoking.[11]

One hopeful conclusion from the CDC study[1] is a reduction of the prevalence of mild-to-moderate COPD in people under age 55. If we can reduce teenage smoking and find the middle-age smokers with incipient stages of disease, we may be able to stop the upward trend of hospitalization and mortality.

It is time for a call to arms. Early identification and treatment of COPD can make an impact. Smoking cessation and the use of a growing number of bronchoactive drugs can improve the outcome of COPD. We can prevent or forestall the progress into the advanced stages of the disease. "*Test Your Lungs, Know Your Numbers,*" stay healthy, and enjoy life in a smoke-free environment.

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# Original Contributions

## Performance of a New Screening Spirometer at a Community Health Fair

Robin J Schoh MSc MBA, Laura J Fero RN, Howard Shapiro PhD, Jordan P Aslor MBA RPFT, Oscar J Kaelin MSEE, Donald R Rollins MD, and Thomas L Petty MD

**OBJECTIVE: Compare the results from a new screening spirometer (EasyOne) with the results from a standard laboratory spirometer (Vmax) approved by the American Thoracic Society. SETTING: A health fair at a community hospital. METHODS: We measured forced expiratory volume in the first second ($FEV_1$) and forced expiratory volume in the first 6 seconds ($FEV_6$). With the screening spirometer, good quality testing was achieved in 359 of 394 subjects (91%), and 115 subjects were also tested with the standard laboratory spirometer. The best test values for $FEV_1$ and $FEV_6$ were taken for 3 tests that agreed within 3%. $FEV_6$ was extrapolated from forced vital capacity on the printouts from the standard laboratory spirometer. RESULTS: Correlations between the screening spirometer results and the standard laboratory spirometer were excellent for $FEV_1$ ($r = 0.93$), $FEV_6$ ($r = 0.96$), and $FEV_1/FEV_6$ ($r = 0.72$) ($p = 0.001$ for all comparisons). The 95% limits of agreement (mean difference between the 2 spirometers ± 1.96 standard deviations) were: −0.18 and 0.69 for $FEV_1$; −0.24 and 0.81 for $FEV_6$; and −0.12 and 0.13 for $FEV_1/FEV_6$. CONCLUSION: The new screening spirometer is suitable for clinical use.** *Key words: screening, spirometry, spirometer, EasyOne, SensorMedics, Vmax, health fair.* [Respir Care 2002;47(10):1150–1157]

### Introduction

Screening for occult disease is commonplace in the United States.[1,2] Community-sponsored health and wellness programs often offer tests for blood pressure, eyesight, hearing, cholesterol, diabetes, osteoporosis, breast, colon, and prostate cancer, and tests for less common disorders. Such screening is often conducted at community health fairs. Spirometry screening is sometimes done at such events.

Since spirometry is predictive of risk of death from heart attack, stroke, lung cancer, chronic obstructive pulmonary disease, and all-cause mortality, it is an appropriate addition to community health screening projects. One previous community project offered spirometric screening to the entire metropolitan population of Denver, Colorado, on a single day.[3] In fact, 2,586 patients were tested at multiple locations during two 4-hour periods on 2 separate Saturday mornings. Follow-up showed that such screening, using a new and previously validated electric spirometer,[4] yielded credible results.[3]

See the Related Editorial on Page 1145

A new national health care initiative recommends routine spirometry screening of all smokers over age 45 and of anyone with chronic cough, mucus hypersecretion, dys-

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Fig. 1. Participants at the Loveland Health Fair using the EasyOne screening spirometer. The spirometry was conducted by nurses and respiratory therapists.

pnea on exertion, or wheeze.[5] That initiative is called the National Lung Health Education Program (NLHEP).[6]

In response to the NLHEP several spirometer manufacturers have developed and marketed new, simple, accurate, and inexpensive hand-held spirometers for the purpose of promoting widespread spirometric screening. One such new device (EasyOne, ndd Medical Technologies, Andover, Massachusetts) meets American Thoracic Society standards and has United States Food and Drug Administration approval. We used several EasyOne spirometers at a Saturday-morning community health fair (2 sessions, 4 weeks apart). We aimed to validate the performance of the new spirometer and its agreement with an established American Thoracic Society-approved spirometer (Vmax, VIASYS Healthcare/SensorMedics, Yorba Linda, California) that is commonly used in pulmonary function laboratories today.

Table 1. Demographics of Health Fair Participants

| | |
|---|---|
| $n$ | 359 |
| Mean age (y ± SD) | 60.8 ± 12.9 |
| Age range (y) | 23–91 |
| Female | 236 (66%) |
| Male | 123 (34%) |

The purpose of this report is to document the performance of the EasyOne device and its agreement with the Vmax spirometer. A secondary aim was to learn the number of abnormal spirometry measurements detected at such a health fair event. A later study will evaluate whether the finding of new spirometric abnormalities resulted in any behavioral changes in subjects whose spirometry was abnormal.

## Methods

Following 2 briefing sessions with the hospital wellness, community nursing, respiratory therapy, and administrative staffs, and a familiarity program that emphasized the performance characteristics of the new screening spirometer, we conducted spirometry on a convenience sample of participants at a community health fair. Written, informed consent was obtained from all subjects. The study was approved by the Human Subjects Committee of McKee Hospital, Loveland, Colorado. The community health fair took place between 6:30 AM and noon on 2 Saturday mornings, April 1st and April 29th, 2000, at McKee Hospital, Loveland, Colorado, the only community hospital that serves Loveland, which has a population of about 60,000. Participants came by previously arranged appointments. In all, 1,270 people came for the full array of tests offered. Spirometry had not previously been offered at this annual health fair.

Before the spirometry each subject filled out a 1-page history form that focused on common pulmonary symp-

Table 2. Screening Spirometer Test Results

| | | |
|---|---|---|
| Normal | | 257 (72%) |
| Abnormal | | 63 (18%) |
| Obstructive defect | 45 (13%) | |
| Restrictive defect | 18 (5%) | |
| Borderline | | 39 (10%) |
| Total | | 359 (100%) |

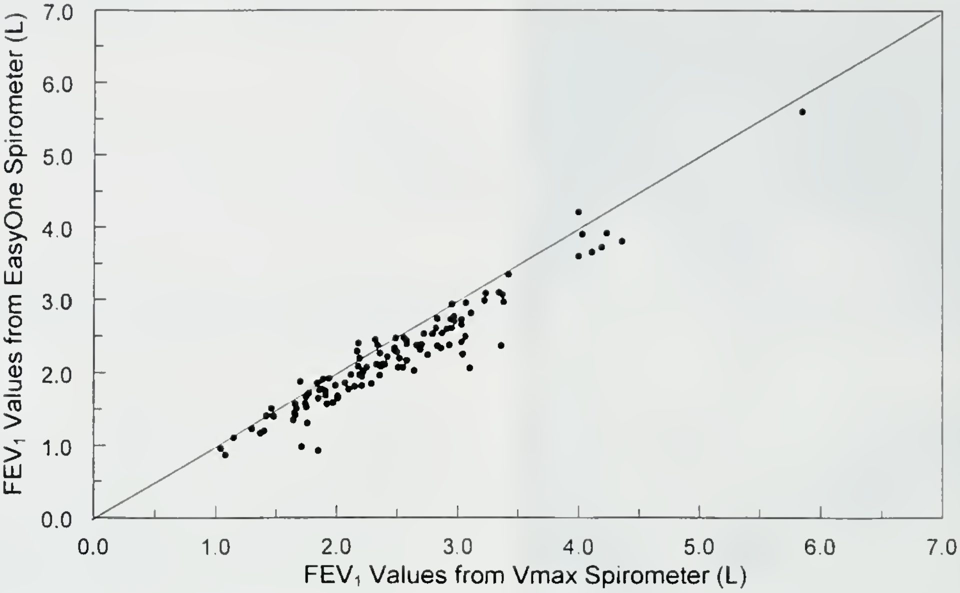

Fig. 2. Forced expiratory volume in the first second ($FEV_1$) values from the EasyOne screening spirometer versus the Vmax spirometer.

toms, past diagnoses of pulmonary disease, smoking history, family history, and occupational history, and asked about age, height, sex, and weight.

Five screening spirometers were used during the first testing day and 7 during the second testing day. All the spirometers were calibrated with a 3 L syringe. Tests with the Vmax device were all conducted by a respiratory therapist with certification in spirometry (ie, a registered pulmonary function technician). The EasyOne devices were used by nurses and other technicians with varied but limited experience in spirometry. These personnel had all received an approximately 1-hour instruction session with the EasyOne. Participant waiting times for testing were estimated to be < 5 min in most cases. Few participants left the waiting queue. Figure 1 illustrates the use of the screening spirometer. All testing was done with the participants in the seated position. Nose clips were not used. A forced vital capacity test of at least 6 seconds was required.

A minimum of 3 tests were done with both the EasyOne and Vmax devices. The best of the tests that agreed within 3% were recorded for forced expiratory volume in the first

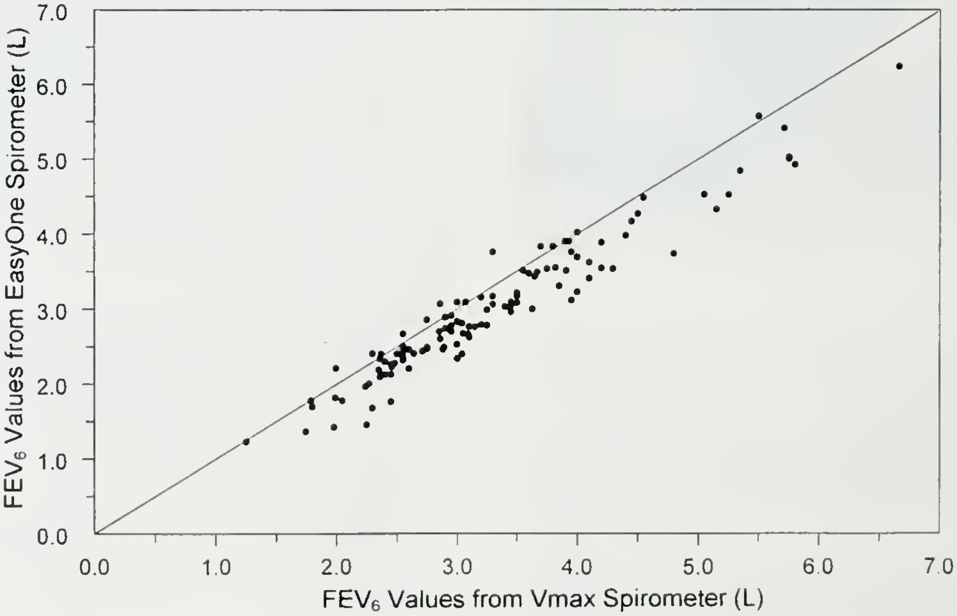

Fig. 3. Forced expiratory volume in the first 6 seconds ($FEV_6$) values from the EasyOne screening spirometer versus the Vmax spirometer.

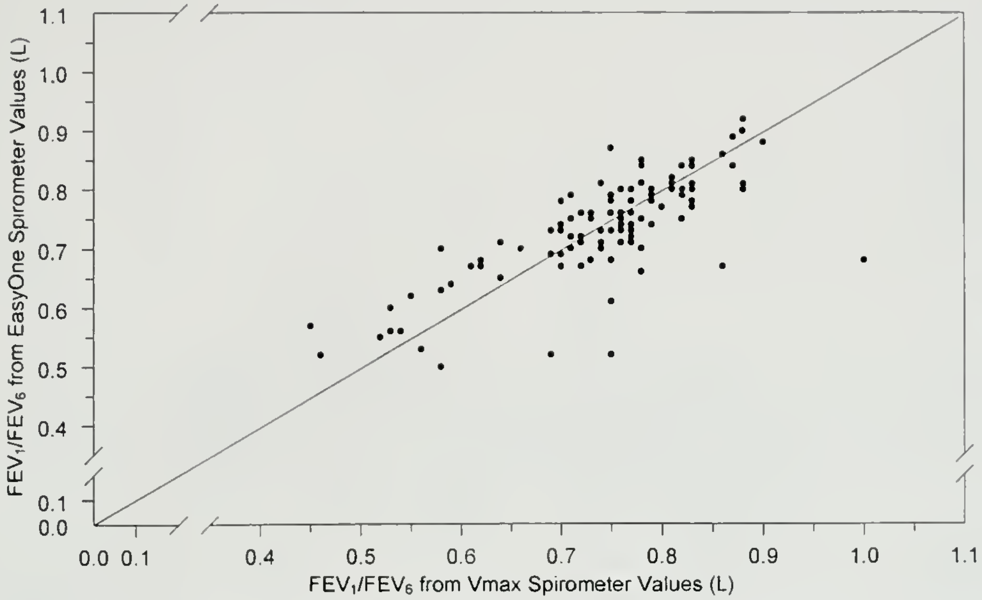

Fig. 4. Comparison of the ratio of forced expiratory volume in the first second ($FEV_1$) to forced expiratory volume in the first 6 seconds ($FEV_6$) from readings from the EasyOne screening spirometer and the Vmax spirometer.

second ($FEV_1$) and forced expiratory volume in the first 6 seconds ($FEV_6$). That criterion was arbitrary but within the American Thoracic Society standard of 200 mL for $FEV_1$ and forced vital capacity for values < 6,000 mL.[7] The device gives an audible cue to continue to exhale for a full 6 seconds.

In an attempt to establish agreement between the results obtained for the EasyOne screening spirometer and the Vmax spirometer, as many patients as possible were encouraged to have "confirmatory testing" following the initial screening spirometry. Enthusiasm for the second testing was higher among those patients who had abnormal test results with the screening spirometer. Additional testing was less popular among subjects who had normal results, because there were other screenings they wanted to do at the health fair. As a result, 115 subjects had both spirometric tests. The $FEV_6$ was extrapolated visually and manually for the Vmax spirometer on each expiratory spirogram. In only 1 instance was the $FEV_6$ not achieved with the Vmax instrument, but a plateau was reached.

A pulmonologist (TLP) was present to answer questions that are common when a newly found abnormality is reported to a participant. All test results were printed and each participant was given the results to keep with his or her personal health care record or to take to his or her personal physician.

The statistical methods we used included descriptive statistics and a measure of agreement between the 2 devices. Agreement was evaluated using 95% limits of agreement (assuming normality), as described by Bland and Altman.[8] Basically, the 95% limits of agreement (mean difference between the 2 devices ± 1.96 standard deviations) are expected to encompass approximately 95% of the population of individual differences. The limits are examined with regard to the clinical acceptance of differences of that magnitude.

## Definition of Abnormal

The presence of an obstructive ventilatory defect was defined as an $FEV_1/FEV_6 < 0.70$ and an absolute $FEV_1$ less than the lower limit of normal. Normal values for $FEV_1$ and $FEV_6$ were obtained from a large population study done as part of the National Health and Nutrition Examination Survey (NHANES III).[9] $FEV_6$ is an accepted surrogate for both obstructive and restrictive ventilatory disorders.[10] A restrictive ventilatory defect was defined as an $FEV_1/FEV_6$ of > 0.80 and an $FEV_6$ less than the lower limit of normal. A borderline abnormality was arbitrarily

Table 3. Mean Differences Between Vmax and EasyOne Readings

| Variable | *n* | Mean Difference (L)* | SD |
|---|---|---|---|
| $FEV_1$ | 113 | 0.254 | 0.221 |
| $FEV_6$ | 112 | 0.288 | 0.267 |
| $FEV_1/FEV_6$ | 113 | 0.00596 | 0.0635 |

*Vmax readings minus EasyOne readings
$FEV_1$ forced expiratory volume in the first second
$FEV_6$ forced expiratory volume in the first 6 seconds

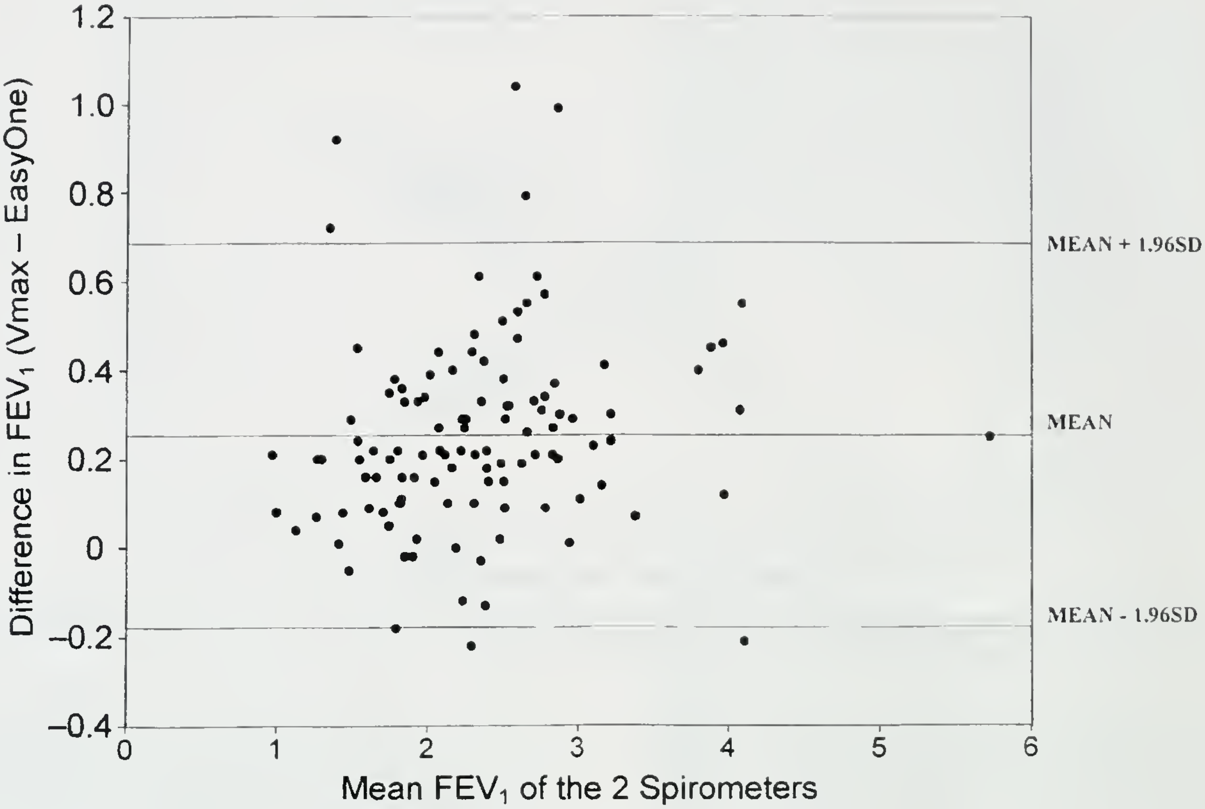

Fig. 5. Difference in forced expiratory volume in the first second ($FEV_1$) between the EasyOne screening spirometer and the Vmax spirometer versus mean $FEV_1$ of the 2 devices.

defined by one of us (TLP) as subjects age ≥ 69 with an absolute $FEV_1$ of 2.5 L and an $FEV_6$ of 3.5 L. This is justified by the fact that very mild abnormalities in older subjects are usually not clinically important.[11]

### Results

In all, 394 subjects were enrolled for screening spirometry (149 on April 1 and 245 on April 29). The higher enrollment on the second testing day was attributed to the use of more screening spirometers (5 on the first day, 7 on the second). On the first day, 1 screening spirometer malfunctioned, reducing the total number of screening spirometry stations to 4. There were no other malfunctions of the screening spirometer. Another factor in the higher enrollment on the second testing day was the use of volunteers to assist the subjects in completing the questionnaire prior to spirometry. Incomplete or inadequate test results (ie, failure to successfully complete 3 satisfactory screening maneuvers) occurred with 35 subjects (9%). Thus, good quality testing was accomplished in 359 subjects (Table 1).

Table 1 shows the demographics of the subjects who had 3 apparently satisfactory tests. Table 2 lists the number of normal and abnormal spirometric tests for the 359 subjects.

### Spirometer Comparisons

When we reviewed all the spirograms, the results of 2 comparisons were considered "outliers" and excluded from the analysis. These results were outside 3 standard deviations. In both subjects the results from the EasyOne screening spirometer were significantly lower than those from the Vmax device: $FEV_6$ 3.50 L versus 2.16 L in one subject, and $FEV_1$ 3.51 L versus 1.16 L in the second subject. Visual review of those spirograms revealed a poor effort on the screening spirometer in each instance.

Figures 2, 3, and 4 display the line of equality and the paired device results for $FEV_1$, $FEV_6$, and the ratio of $FEV_1$ to $FEV_6$. This is a traditional method of comparing values obtained with 2 instruments. It has the advantage of displaying the actual test results. Table 3 shows the mean differences (Vmax minus EasyOne). Figures 2 and 3 suggest that for $FEV_1$ and $FEV_6$ the EasyOne device slightly underestimated the results of the Vmax device, but this may be due to a learning effect, since the Vmax tests always followed the EasyOne tests, for practical and logistical reasons. Also the Vmax technician had more spirometry experience than the personnel who used the EasyOne. The $FEV_1/FEV_6$ ratios show no systematic under-estimation or over-estimation.

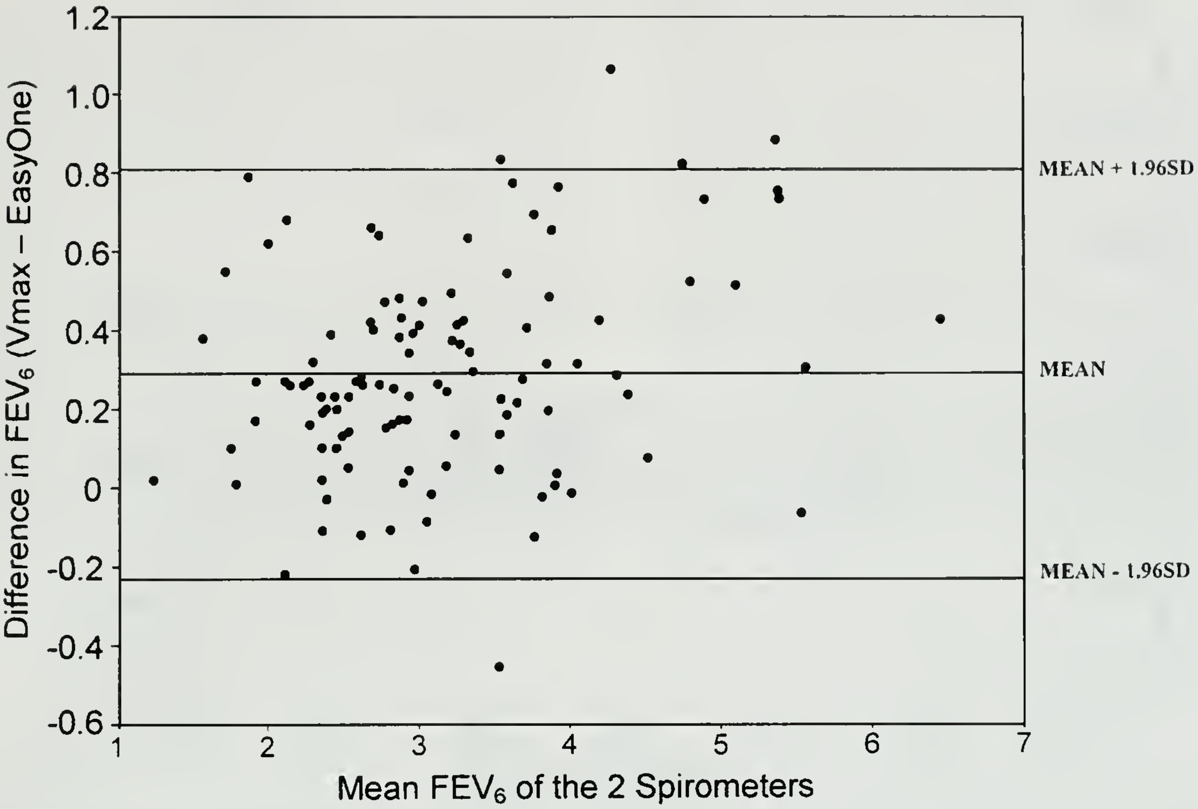

Fig. 6. Difference in forced expiratory volume in the first 6 seconds ($FEV_6$) between the EasyOne screening spirometer and the Vmax spirometer versus mean $FEV_6$ of the 2 devices.

The correlation coefficients of the screening spirometer results and the standard laboratory spirometer were excellent for $FEV_1$ ($r = 0.93$), $FEV_6$ ($r = 0.96$), and $FEV_1/FEV_6$ ($r = 0.72$) ($p = 0.001$ for all comparisons). However, the use of correlation coefficients has been criticized,[8] so we examined the limits of agreement to evaluate the differences between the results obtained by the 2 devices. The limits of agreement were: −0.18 and 0.69 for $FEV_1$; −0.24 and 0.81 for $FEV_6$; and −0.12 and 0.13 for $FEV_1/FEV_6$.

Figures 5, 6, and 7 plot the differences in $FEV_1$ and $FEV_6$ values and the $FEV_1/FEV_6$ ratios between the 2 devices versus the mean $FEV_1$ and $FEV_6$ values and the $FEV_1/FEV_6$ ratios, as suggested by Bland and Altman.[8] Also included are the limits of agreement. Figures 5 and 6 suggest that there is no systematic bias between the 2 devices. Several observations outside of the limits of agreement were investigated and retained as clinically acceptable.

One of the authors (TLP) independently interpreted the spirometry results. If the 39 borderline abnormal results are omitted from the comparisons, the computerized interpretation by the device agreed with the clinician's interpretation on 92% (330/359) of the spirograms.

## Discussion

The NLHEP recommends screening spirometry for all smokers over age 45 and for anyone with chronic cough, mucus hypersecretion, inappropriate dyspnea, or wheeze.[5] Testing at the Loveland Health Fair was not restricted to that population. Anyone desiring spirometry was given at least 3 attempts to produce valid results. The majority of the tests (91%) were of satisfactory quality. The new screening spirometers functioned well throughout the study, with one exception during the first testing session.

The $FEV_1$ and $FEV_6$ values obtained with the EasyOne screening spirometer were in generally good agreement with the Vmax spirometer, which is a commonly used pulmonary function laboratory diagnostic spirometer. A large number of subjects ($n = 359$) were successfully tested during the total of 9 hours of testing over the 2 Saturday morning health fair sessions.

The EasyOne uses the Doppler principle of flow sensing. It is not affected by altitude (the spirometry reported herein was conducted at about 5,000 feet above sea level). A temperature sensor near the mouthpiece corrects ambient-temperature-and-pressure-saturated (ATPS) readings to

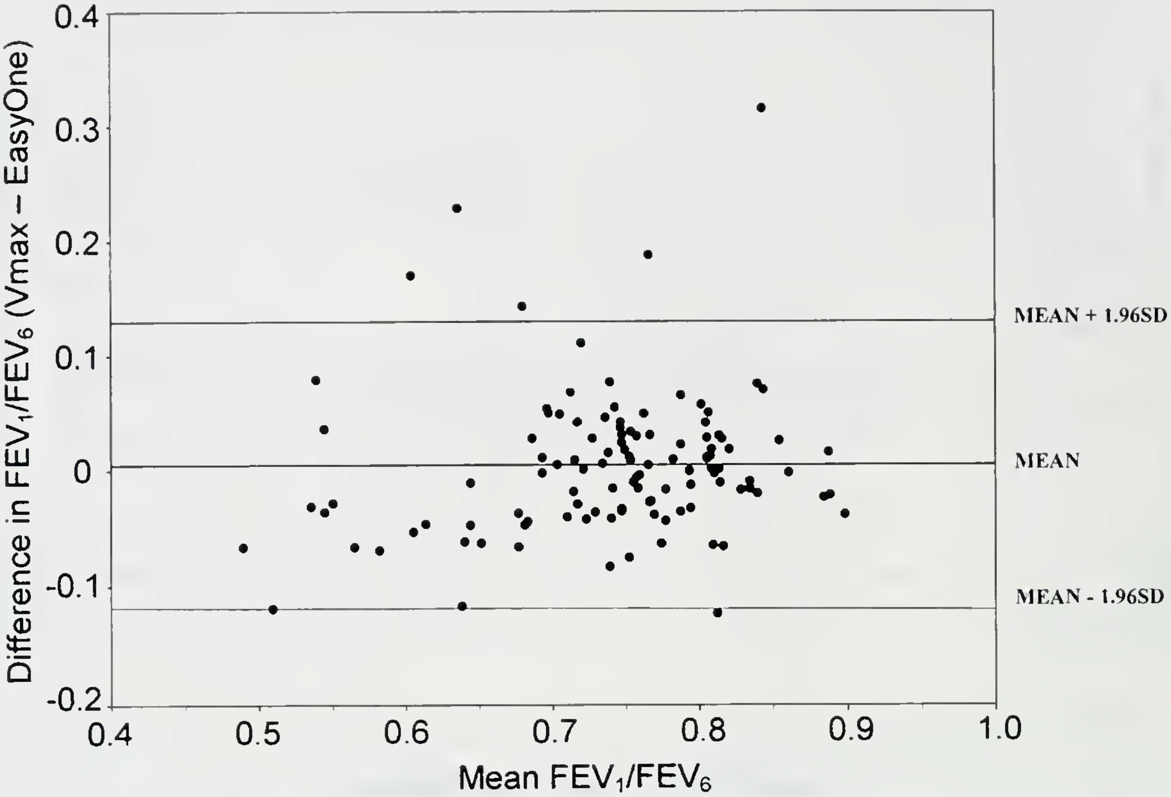

Fig. 7. Difference between the ratios of forced expiratory volume in the first second ($FEV_1$) to forced expiratory volume in the first 6 seconds ($FEV_6$) versus the mean $FEV_1/FEV_6$ ratios from readings from the EasyOne screening spirometer and the Vmax spirometer

body-temperature-and-pressure-saturated (BTPS) readings.

We found 63 (17%) subjects with abnormal spirometry readings, which in a health care setting is consistent with results from a random population.[10] The arbitrary definition of borderline (age ≥ 69 and $FEV_1$ of 2.51 and $FEV_6$ of 3.5) is reasonable. The subjects with only mild abnormalities probably do not need any further evaluation unless they develop symptoms.

It will be important to determine whether knowledge of the spirometric abnormalities discovered at the health fair has caused any behavioral change among those participants or their physicians. Did the participant stop smoking or receive any therapy aimed at improving lung function? These questions are being pursued in an ongoing follow-up study in the community.

The NLHEP Subcommittee on Screening Spirometry recommends validation testing of each new screening device placed on the market following Food and Drug Administration approval. The details of validation recommendations have been published.[5] It is the responsibility of each manufacturer that develops a screening spirometer for widespread application to complete such a validation study before stating that the device meets the NLHEP spirometry recommendations and standards. The health fair study reported herein cannot be considered to have fulfilled those NLHEP recommendations. Our purpose was to gain knowledge about the device's health fair performance and to conduct a preliminary evaluation of its accuracy compared with an established laboratory spirometer.

Another chronic obstructive pulmonary disease initiative is known as the Global Initiative for Chronic Obstructive Lung Disease (GOLD), which is an international organization sponsored by the World Health Organization and the United States National Heart, Lung, and Blood Institute. GOLD recommends the development and testing of simple, user-friendly spirometers for widespread clinical use.[12] "Spirometers need to be developed that can ensure economical and accurate performance when a relatively untrained operator administers the test."[12]

## Conclusions

The results of this study suggest that the EasyOne spirometer performed satisfactorily in a health fair setting. The agreement between the readings of the EasyOne and the Vmax devices was acceptable for clinical purposes.

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# A Dyspnea Evaluation Protocol for Respiratory Therapists: A Feasibility Study

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Daniel Reily RRT, and Barry D Fuchs MD

**PURPOSE: We tested the feasibility of incorporating a dyspnea evaluation protocol into bedside assessments routinely performed by respiratory therapists (RTs) on mechanically ventilated patients at a university teaching hospital. METHODS: A dyspnea assessment protocol was incorporated into the RT assessments performed at 4-hour intervals on endotracheally intubated, mechanically ventilated patients in our medical and surgical intensive care units. RTs were asked to inquire of all responsive patients: "Are you feeling short of breath right now?" and, if yes, "Is your shortness of breath mild, moderate, or severe?" We analyzed 324 consecutive patient ventilator flow sheets from 77 medical and 161 surgical intensive care unit patients. RESULTS: Dyspnea scores were recorded during 1,870 of 2,539 scheduled RT patient assessments. The protocol compliance rate was 74%. Patients were sufficiently responsive to answer the protocol questions during 32.1% of the bedside assessments. Dyspnea was recorded in 11% (67/600) of those encounters. Dyspnea was described most often as mild. CONCLUSIONS: Initial implementation of a dyspnea evaluation protocol was moderately successful in prompting RTs to ask mechanically ventilated patients whether they felt short of breath during scheduled bedside visits. A rapid bedside evaluation for dyspnea may prove useful in evaluating the effect on patient distress of implementing protocols designed to optimize ventilator settings or the use of sedating drugs during mechanical ventilation. By this approach RTs may also be able to promote a patient-centered approach to managing respiratory failure in the intensive care unit.**

*Key words: dyspnea, dyspnea evaluation protocol, shortness of breath scale, mechanical ventilation, respiratory therapists.* [Respir Care 2002;47(10):1158–1161]

## Introduction

Even when adequate arterial blood gas values and acid-base balance are restored, mechanical ventilation does not invariably alleviate dyspnea experienced by patients in respiratory failure.[1–4] Because they are endotracheally intubated, mechanically ventilated patients are unable to [2]speak and therefore cannot complain about shortness of breath or other forms of distress unless asked specifically.[5] Although mechanically ventilated patients often appear apprehensive or resistive when short of breath,[1,5] there are no physical findings associated specifically with dyspnea. Thus, shortness of breath can develop or become worse during mechanical ventilation without the awareness of bedside caregivers.

Clinical investigators have begun to study the prevalence and severity of dyspnea experienced by patients undergoing mechanical ventilation, using symptom assessment scales[6] or numerical or visual analogue scales.[3,7]

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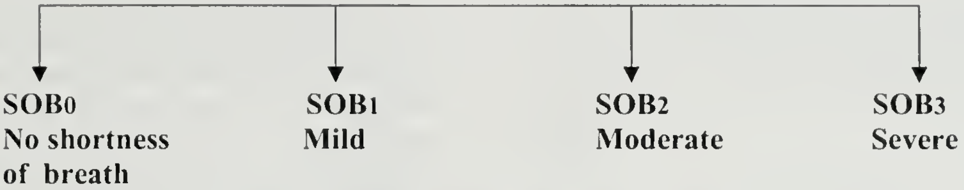

Fig. 1. Shortness of breath (SOB) evaluation scale.

Published studies have shown that these scales can serve as useful tools for quantifying dyspnea during the progress of respiratory failure and in response to therapy.[1–4,6–8]

The observation that dyspnea in mechanically ventilated patients can be evaluated quantitatively raises the question of whether systematic dyspnea assessment can facilitate clinical quality improvement efforts and thus improve regular patient care. Nurses at many hospitals now routinely assess mechanically ventilated patients for the presence and severity of pain, especially after trauma or surgery. Might similar approaches also be employed to detect and treat shortness of breath during mechanical ventilation? If so, respiratory therapists (RTs) may be ideally suited to conduct those assessments. RTs routinely perform and record detailed physiologic assessments of mechanically ventilated patients, at frequent intervals throughout the day and night. Along with the assessment of the ventilator settings and alarms it is also important to assess the patient-ventilator interface. An evaluation of patient dyspnea is essential in that assessment. Addition of a brief dyspnea assessment to physiologic evaluations might add little time to regularly scheduled bedside visits. Therapists are suitably trained to evaluate dyspnea in the context of the other information on respiratory and ventilator function obtained during their visits. RTs may also be able to alleviate dyspnea directly in some instances by such interventions as adjusting the ventilator,[9,10] suctioning the airway, or administering a bronchodilator.

In this study we sought to evaluate the feasibility of adding a brief dyspnea evaluation protocol to routine bedside assessments of mechanically ventilated patients in the medical and surgical intensive care units (ICUs) of a university teaching hospital by measuring the average time required to complete the protocol and the compliance rate of RTs assigned to use the protocol. This study also afforded an opportunity to estimate the prevalence of dyspnea in the diverse population of patients undergoing mechanical ventilation at our hospital.

## Methods

A dyspnea assessment protocol was devised using 2 standardized questions: (1) "Are you feeling short of breath right now?" and, if yes, (2) "Is your shortness of breath mild, moderate, or severe?" Both questions could be answered by a nod of the head. We inquired about shortness of breath "right now" because many critically ill patients experience short-term memory deficits that interfere with assessment of dyspnea over time. To minimize the time required for assessment, and because our interest is in improving routine patient care rather than in measuring fine gradations in the severity of dyspnea, we used a simplified, 3-point intensity scale (mild, moderate, severe) rather than a 10-point numerical or a visual analogue scale. Responses to the 2 questions were reported using 1 of 4 abbreviated designations, as shown in Figure 1. At the time of assessment the appropriate designation was recorded on a ventilator flow sheet that was modified to include a column for dyspnea assessment, in addition to the usual physiologic data and ventilator settings.

The respiratory therapy staff received from one of us (BDF) standardized education on protocol methodology. The rational for dyspnea assessment and the purpose of the study were described in detail to the participating RTs. No special inducements or rewards were offered to improve compliance with the protocol. Beginning in January 2001, RTs working on all 3 daily shifts were expected to perform and record dyspnea assessments during routine bedside visits at 4-hour intervals on all mechanically ventilated patients in the ICUs at the Hospital of the University of Pennsylvania.

We analyzed the first 324 consecutive patient ventilator flow sheets completed following implementation of the protocol. There were 155 sheets from 77 medical ICU patients and 169 sheets from 161 surgical ICU patients. Our medical ICU is a 12-bed unit that serves critically ill patients transferred from other hospitals and recipients of solid organ and bone marrow transplants, in addition to patients admitted through an inner-city emergency department. Our 55-bed surgical ICU serves critically ill surgical patients in the perioperative period. The patient population includes cardiothoracic, general surgical, neurosurgical, trauma, vascular, and surgical subspecialty patients. All study patients were receiving mechanical ventilation by endotracheal intubation or tracheostomy. All study subjects spoke English and were $\geq$ 18 years of age.

In a separate analysis we measured the mean time required to complete the dyspnea assessment during 10 routinely scheduled bedside visits to different patients by 5 RTs who were experienced with application of the protocol.

Table 1. Summary Data of All Dyspnea Assessments

| | |
|---|---|
| Scheduled respiratory therapy patient assessments | 2,539 |
| Scores recorded | 1,870 (73.7%) |
| Blank | 669 (26.3%) |
| Assessable patients | 600 (32.1%) |
| Not assessable | 1,270 (67.9%) |
| SOB0 | 533 (89%) |
| SOB1 | 47 (7.8%) |
| SOB2 | 12 (2%) |
| SOB3 | 8 (1.3%) |

SOB = shortness of breath scale

## Results

A total of 324 patient ventilator sheets from 238 patients, totaling 2,539 scheduled dyspnea assessments (Table 1) were evaluated. RT protocol compliance was 74% (1,870/2,539). Six-hundred of 1,870 assessments (32.1%) recorded a score of dyspnea. The rest of the patients were not assessable because they were not alert or communicative, as a result of central nervous system dysfunction or sedation. Shortness of breath (SOB1, 2, or 3) was recorded in 11% (67/600) of the dyspnea assessments performed. Shortness of breath was graded as mild in 7.8% (47/600) of the assessments, as moderate in 2% (12/600), and as severe in 1.3% (8/600).

The data from the medical and surgical ICU populations were analyzed separately (data not shown). There was no difference in the number of assessable evaluations in the 2 units (by *t* test, $p = 1.0$). Nor was there a difference in the prevalence and severity of dyspnea (by *t* test, $p = 0.8$).

The mean ± SD time required to complete the dyspnea evaluation protocol was 17 ± 11 s during 10 routine bedside visits by 5 experienced RTs.

## Discussion

This study demonstrates that a simple method of dyspnea assessment can be incorporated into the routine bedside assessments performed by RTs. Experienced RTs required an average of 17 seconds to complete the dyspnea assessment protocol we developed for this study. Shortly after introduction of the new protocol, RT compliance was moderately good overall (74%) and was similar in the medical and surgical ICUs.

This is the first study to estimate the prevalence and severity of dyspnea across the spectrum of endotracheally intubated, mechanically ventilated ICU patients at 1 hospital. Approximately one third of study subjects at our regional referral hospital were able to answer the 2 yes-or-no questions during routine bedside assessments by RTs. The others were deeply sedated or too impaired neurologically to respond. Among patients who could answer the protocol questions, RTs identified dyspnea during 11% of bedside visits. Most often the dyspnea was described as mild, but some patients described their dyspnea as moderate or severe.

Although nurses are encouraged to frequently evaluate critically ill patients for discomfort and distress, the effectiveness of those assessments for detecting dyspnea in routine practice is unknown. Physical agitation, anxiety, and other nonspecific signs of respiratory distress during mechanical ventilation may be erroneously attributed to anxiety and treated accordingly. As a result, the duration of mechanical ventilation may be unnecessarily prolonged in some instances by heavy use of sedating medications to treat underlying respiratory distress that could be alleviated directly if recognized.[11–13]

Even if bedside nurses are often effective at identifying dyspnea, additional routine dyspnea assessment by RTs may improve patient care in several ways. First, the more frequently dyspnea assessments are performed the less time is likely to elapse between onset and discovery of this distressing symptom. Second, RTs are well trained to relieve dyspnea by optimizing ventilator settings,[14] suctioning the airways, or administering bronchodilators. When appropriate, those specific dyspnea treatments are greatly preferable to administration of additional opioids or sedating drugs. Third, routine dyspnea assessment by RTs can help ensure that application of new treatment protocols does not unnecessarily increase patient distress.

Widespread adoption by RTs of a dyspnea assessment protocol may have even broader implications for the practice of respiratory care. To date, RTs and other critical care professionals have focused primary attention on maximizing the short-term survival of their patients. Though much work remains to be done toward that primary objective, recent progress in the treatment of respiratory failure also provides new opportunities to improve the comfort of patients undergoing mechanical ventilation. RTs now manage a number of therapeutic options for patients in respiratory failure, including a variety of ventilation modes and noninvasive mechanical ventilation. By combining routine assessment of respiratory distress with judicious use of those therapies, RTs can expand ICU bedside practice beyond optimizing respiratory mechanical function and gas exchange to include alleviating previously unrecognized patient distress. This expanded approach to individualizing ventilatory support has been called "patient-centered mechanical ventilation."[5]

There are important limitations to the present preliminary study. We did not characterize the medical conditions or characteristics of our study population, which limits the generalizability of the findings. We did not systematically interview participating RTs to determine their acceptance of the protocol, although informal feedback has generally been positive. We did not examine the durability of protocol compliance. Clinical protocols often fare best early after implementation, when attention is focused on maximizing compliance. Long-term compliance will depend on the staff's perception of the protocol's utility, which remains to be tested.

Another limitation is the possibility that an alert patient could be confused and give RTs misleading answers to dyspnea questions. A more detailed patient interview would be required to rule that out, which would impact the feasibility of the protocol. Routine collaboration with the bedside nurse regarding the patient's mental status could minimize the frequency of unreliable responses but would not eliminate the possibility.

Finally, recurrent assessments might cause a patient to intentionally falsely deny feeling short of breath, in order to avoid airway suctioning or other unpleasant interventions, if he or she experienced discomfort following a prior admission of dyspnea to the RT. Although that seems like an uncommon scenario, it would impact the validity of our results and lead us to underestimate the prevalence of dyspnea.

With the goal of maximizing protocol compliance, we used a quick, simple, 3-point dyspnea severity scale (mild, moderate, severe) rather than one of the numerical or visual analogue scales developed by others for research application. It will be useful to compare our scale with those numerical and visual analogue scales, both at baseline and for assessing changes in the severity of dyspnea in individuals and between groups. Finally and most importantly, if routine RT dyspnea evaluation of mechanically ventilated patients proves generally feasible, much additional research will be needed to develop strategies for minimizing dyspnea without compromising outcomes in the management of respiratory failure.

## Conclusions

Implementation of a hospital-wide dyspnea evaluation protocol into routine RT assessments of mechanically ventilated patients was feasible and effective. RTs identified dyspnea among a minority of the mechanically ventilated patients. Although most of those patients reported their dyspnea to be mild, others were found, unexpectedly, to have moderate or severe dyspnea. Routine RT evaluations for dyspnea may prove useful in evaluating the effect on patient distress of implementing protocols designed to optimize ventilator settings or the use of sedating drugs during mechanical ventilation. RTs can thus use a patient-centered approach to managing mechanical ventilation in the ICU.

## ACKNOWLEDGMENTS

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# A Comparison of Intrapulmonary Percussive Ventilation and Conventional Chest Physiotherapy for the Treatment of Atelectasis in the Pediatric Patient

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**OBJECTIVE: Compare intrapulmonary percussive ventilation (IPV) to conventional chest physiotherapy (CPT) and determine their effects on improving atelectasis and static compliance in pediatric patients. METHODS: We conducted a retrospective study of 46 patients who received IPV therapy with the Percussionator IPV-1 ventilator at frequencies of 180–220 cycles/min and pressures of 15–30 cm $H_2O$. Medicated aerosol therapy with albuterol 2.5 mg in 6 mL normal saline solution was delivered with each IPV treatment. Baseline and subsequent chest radiographs were evaluated by a pediatric radiologist. We used an ordinal scoring system to measure the degree of atelectasis to evaluate chest radiographs (4 = complete collapse, 0 = complete resolution). Then we conducted a prospective, randomized, controlled study of intubated and mechanically ventilated patients to compare changes in atelectasis and static compliance. Baseline and daily chest radiographs were evaluated using the same scoring system as in the retrospective pilot evaluation. Patients were ventilated in the volume-controlled, synchronized intermittent mandatory ventilation mode, with tidal volumes of 6–10 mL/kg. Patients were randomized to CPT (clapping and vibration) or IPV at frequencies of 180–220 cycles/min and pressures of 15–30 cm $H_2O$ (equal to the peak pressures on the ventilator), with 6 mL of normal saline solution via medicated aerosol. Both treatments were given every 4 h and lasted 10–15 min. Static compliance measurements were calculated from exhaled tidal volumes and plateau pressures. RESULTS: In the retrospective study the median age of patients receiving IPV was 4.2 years and the median duration of IPV was 6.2 days. A change in atelectasis score from 3 to 1 ($p < 0.001$) was seen. In the randomized, controlled trial the median age of patients was 3.1 years. Atelectasis scores before treatment were comparable between the CPT and IPV groups (median 2.0 for both groups, p = 0.530). Atelectasis scores after treatment were unchanged in the CPT group (median 2.0, p = 0.421) but improved in the IPV group (median 1.0, p = 0.026). Treatment lasted an average of 6.2 days in the CPT group and 2.1 days in the IPV group (p = 0.018). Neither group showed any change in static compliance following treatment. CONCLUSIONS: In the retrospective study a clinically important improvement in atelectasis was seen in patients who received IPV therapy. In the controlled, clinical trial the IPV group showed more clinically important improvement in atelectasis than the CPT group. IPV is a safe and effective method of alternative airway clearance and can be used on patients with artificial airways.** *Key words: intrapulmonary percussive ventilation, chest physiotherapy, atelectasis, pediatric.* [Respir Care 2002;47(10):1162–1167]

## Introduction

Airway clearance modalities are used to increase the effectiveness of cough, assist in mobilizing secretions, resolve atelectasis, and improve ventilation and oxygenation.[1] Conventional chest physiotherapy (CPT) methods include clapping, vibration, and postural drainage, which promote mobilization of secretions and improve cough in patients with atelectasis. Clinical practice guidelines have been es-

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tablished for CPT and positive expiratory pressure (PEP). Oscillatory PEP (using either the Flutter valve or the Acapella device), high-frequency chest wall compression (HFCWC), and intrapulmonary percussive ventilation (IPV) are newer therapies awaiting the development of clinical practice guidelines.[2,3] Pediatric applications of airway clearance therapies include all of the currently established adult modalities. Selection of appropriate therapy is based on the patient's clinical presentation, the indications for treatment, and the patient's ability to perform the therapy.

IPV is the delivery of high frequency, low-volume, positive-pressure breaths in the range of 100–300 cycles/min. This mode of CPT creates an internal percussion effect on the lungs as they are held in the state of partial inspiration.[4] IPV is administered with the Intrapulmonary Percussionator IPV-1 ventilator (Percussionaire, Sandpoint, Idaho) via mouthpiece, mask, or artificial airway. Early experiences with IPV for cystic fibrosis and chronic obstructive lung disease demonstrated effective secretion mobilization, improved atelectasis, and enhanced oxygenation.[1] IPV was introduced in the mid-1980s as an airway clearance modality and an adjunct to standard practice with adults. It entered pediatric practice in the 1990s. To date there have been no safety and efficacy studies of IPV for airway clearance in intubated and mechanically ventilated pediatric patients.

## Methods

We conducted 2 studies using IPV with pediatric patients. The first study was a retrospective evaluation to determine if IPV showed any radiographic evidence of clinical improvement of atelectasis or had adverse effects. Positive results from the retrospective study led to a randomized, controlled trial comparing the effects of IPV to standard CPT and postural drainage. We hypothesized that IPV would reduce atelectasis and improve static compliance.

### Retrospective Study

This study evaluated pediatric patients in the Rainbow Babies and Children's Hospital pediatric intensive care unit, rehabilitation unit, and acute care areas. We studied patients who had radiographic evidence of atelectasis. IPV was ordered at the physician's discretion as an alternative to CPT. Patients receiving IPV were assigned a baseline "atelectasis score" (Table 1). Atelectasis was characterized by collapse of lung segments. Collapse was identified by the presence of at least 1 of the following: mediastinal shift toward the affected side, elevation of the hemidiaphragm on the affected side, identification of the interlobar fissure on the affected side, and (in most severe cases) reduction of intercostal spaces on the affected side.

Table 1. Atelectasis Scoring System

| Score | Description |
|---|---|
| 0 | Complete resolution of collapse |
| 1 | Partial collapse of 1 segment or lobe |
| 2 | Partial collapse of ≥ 2 segments or lobes |
| 3 | Complete collapse of 1 segment or lobe |
| 4 | Complete collapse of ≥ 2 segments or lobes |

Partial collapse was defined as linear densities extending from the mediastinum without shift, representing segmental collapse. Complete collapse was defined as presence of mediastinal shift toward the collapse, with elevation of the hemidiaphragm and the presence of air bronchograms on the affected side.

Patients received IPV under the direction of a physician. IPV was administered with a Percussionator IPV-1 ventilator at frequencies ranging from 180–220 cycles/min and pressures of 15–30 cm $H_2O$. An aerosol consisting of 2.5 mg albuterol and 6 mL of normal saline solution was nebulized with each treatment. Treatments were administered every 4–6 h, as ordered by the physician. The duration of treatment was determined by the amount of time required for the medication to be nebulized, usually about 10 min. Atelectasis scores were obtained upon reevaluation of daily chest radiographs.

### Randomized Controlled Trial

The follow-up study was a prospective, randomized, controlled comparison of CPT and IPV administered for the treatment of atelectasis in a group of intubated and mechanically ventilated pediatric patients. The study protocol was approved by the hospital institutional review board, and parental consent was obtained prior to randomization or initiation into the study. Entry criteria included:

- Intubation and mechanical ventilation in the pediatric intensive care unit
- Evidence of atelectasis on chest radiograph
- A minimum patient weight of 3 kg

We arrived at the 3 kg minimum from the retrospective evaluation, in which 2 patients < 3 kg experienced hypotension during the IPV treatments.

Excluded from the study were patients who were febrile, who had secretion cultures positive for bacteria, who had pulmonary air leak, or who had other pulmonary diseases accounting for infiltrates (eg, pneumonia). Subjects were randomized to CPT or IPV by drawing sealed envelopes containing the treatment type, which created an equal and independent chance of being selected to one or the other treatment.[5]

The experimental protocol was initiated following randomization. Baseline data, including blood oxygen saturation (measured via pulse oximetry), blood pressure, breath sounds, and respiratory rate, were recorded before and after each treatment. Routine daily chest radiographs were obtained and atelectasis scores (using the ordinal radiology score system used in the pilot study) were assigned daily by a pediatric radiologist in consultation with a pediatric intensive care physician, both blinded to the type of treatment the patient received. All of the randomized patients were maintained on a Servo 900C ventilator (Siemens, Danvers, Massachusetts) for the duration of the study.

The following ventilation parameters were maintained during the study period: volume-controlled, synchronized intermittent mandatory ventilation; positive end-expiratory pressure 5 cm $H_2O$; tidal volume 6–10 mL/kg; and respiratory frequency determined by the patient's age and underlying clinical condition. Exhaled tidal volume and plateau pressures were measured with a respiratory profile monitor ($CO_2$SMO Plus! Novametrix Medical Systems, Wallingford, Connecticut), and static compliance was calculated from those values, as follows:

$$C_{stat} = \frac{V_T(mL)}{P_{plat}(cm\ H_2O) - PEEP(cm\ H_2O)}$$

in which $C_{stat}$ is static compliance, $V_T$ is tidal volume, $P_{plat}$ is plateau pressure, and PEEP is positive end-expiratory pressure. Plateau pressure was measured by depressing and holding the inspiratory pause button on the Servo 900C at end inspiration.

### Therapeutic Modalities

Patients randomized to conventional treatment received CPT for 10–15 min every 4 hours, administered by a respiratory therapist. CPT consisted of percussion, clapping, and vibration over areas of atelectasis. All patients were suctioned at the completion of each treatment.

Patients randomized to IPV received treatments every 4 h. The treatment involved removing the Servo 900C ventilator circuit tubing from the endotracheal tube adapter and attaching the IPV machine's tubing to the endotracheal tube adapter. During IPV, patients were maintained in the supine position. Treatment settings were determined prior to initiation of treatment. IPV pressure settings were set equal to the peak pressures observed during routine mechanical ventilation (15–30 cm $H_2O$). The frequency was determined by adjusting the impact control knob to a corresponding frequency that was manually counted at 180–220 cycles/min. IPV treatments were given with 6 mL of normal saline solution and lasted 10 min.

Although bronchodilators are known to increase airway caliber and cilia beat frequency and to enhance mucus clearance, they were not used in this study. Bronchodilators were avoided to allow objective evaluation of the effectiveness of the therapy, without possibly confounding results by the administration of medication. Upon initiating therapy, chest rise was observed and breath sounds were assessed. IPV intervals lasted 20 s, followed by 5–10 s pauses. Additional pauses were interspersed when the patient needed suctioning or during episodes of coughing.

Following both types of treatment, vital signs, treatment variables, static compliance, and adverse reactions were recorded. Patients exited the study when atelectasis had resolved on chest radiograph (as indicated by an atelectasis score of zero) or on extubation.

The Mann-Whitney rank sum test was used to compare the scores from before and after treatment, and differences were considered statistically significant when $p \leq 0.05$.

## Results

### Retrospective Study

Forty-six patients were evaluated in the retrospective study, ranging in age from 1 month to 15 years (median age 4.2 y). Forty-one patients (90%) received IPV treatments through the artificial airway. Five patients (10%) received IPV via mask. A significant improvement in atelectasis score was seen (from 3 to 1, $p < 0.001$). The median duration of treatment in this study was 6.2 days. No adverse effects were detected from the IPV treatment or from the administration of bronchodilators.

### Randomized Controlled Trial

The randomized, controlled study enrolled 12 participants (5 in the CPT group, 7 in the IPV group), with ages ranging from 7 weeks to 14 years (Table 2). The endotracheal tube sizes used in the study participants ranged from 3.0 to 7.0 mm internal diameter, with 4.0 being the most prevalent. The CPT group showed no change in atelectasis score with treatment ($p = 0.421$), but the IPV group showed improvement, from 2.3 to 0.9 ($p = 0.026$). The duration of treatment to the resolution of atelectasis was significantly less in the IPV group (3.1 vs 6.2 d, $p = 0.018$). There were no significant differences in static compliance, saturation, or respiratory rate with treatment. Neither group experienced any adverse effects as a result of the treatments.

## Discussion

A healthy individual accomplishes airway clearance through mucociliary action and effective cough. Inherent airway clearance mechanisms are efficient under normal

Table 2. Raw Data

| Patient | Atelectasis Score | | Static Compliance (mL/cm $H_2O$) | | $S_{pO_2}$ (%) | | f (breaths/min) | | Treatment Duration (d) | Weight (kg) | Age |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Before* | After | Before | After | Before | After | Before | After | | | |
| CPT 1 | 2 | 2 | 1.9 | 1.9 | 93 | 95 | 44 | 52 | 5 | 4.5 | 4 mo |
| CPT 2 | 2 | 2 | 2.1 | 1.5 | 92 | 93 | 36 | 32 | 7 | 3 | 2 mo |
| CPT 3 | 2 | 2 | 2.4 | 3 | 95 | 95 | 42 | 48 | 8 | 5 | 3.5 mo |
| CPT 4 | 1 | 4 | 36.4 | 34.6 | 91 | 92 | 18 | 14 | 4 | 56 | 14 y |
| CPT 5 | 3 | 3 | 7 | 8.3 | 93 | 93 | 24 | 28 | 7 | 16 | 3 y |
| CPT Mean | 2.0 | 2.6 | 10.0 | 9.9 | 92.8 | 93.6 | 32.8 | 34.8 | 6.2 | 16.9 | – |
| IPV 1 | 3 | 1 | 2 | 2.8 | 91 | 92 | 36 | 33 | 2 | 3 | 3 mo |
| IPV 2 | 2 | 0 | 3.5 | 4 | 93 | 93 | 26 | 28 | 2 | 10 | 22 mo |
| IPV 3 | 3 | 3 | 2.3 | 3 | 92 | 95 | 44 | 42 | 4 | 4 | 4 mo |
| IPV 4 | 1 | 0 | 1.3 | 1.6 | 94 | 95 | 38 | 36 | 2 | 3.8 | 7 wk |
| IPV 5 | 2 | 1 | 7.6 | 6.6 | 93 | 99 | 26 | 28 | 3 | 7.8 | 14 mo |
| IPV 6 | 2 | 1 | 6.5 | 7.2 | 90 | 93 | 36 | 33 | 2 | 8 | 18 mo |
| IPV 7 | 3 | 0 | 7.7 | 8.3 | 94 | 94 | 24 | 25 | 7 | 16 | 3 y |
| IPV Mean | 2.3 | 0.9 | 4.4 | 4.8 | 92.4 | 94.4 | 32.9 | 32.1 | 3.1 | 7.5 | – |

f = respiratory rate

*Values labeled "Before" were obtained after the first treatment. Values labeled "After" were obtained after the last treatment, when the patient exited the study.

$S_{pO_2}$ = oxygen saturation measured via pulse oximetry

CPT = chest physiotherapy

IPV = intrapulmonary percussive ventilation

conditions. Mucus movement is accomplished by the mucociliary escalator, which propels mucus from deep in the lung toward the large airways.[6] Mucus is expelled from the airway by swallowing or cough.

Abnormal physical conditions such as primary respiratory muscle weakness, physical deformities of the chest wall found in restrictive lung disease, genetic multisystem disorders with primary cilia defects, or the presence of atelectasis caused by mucus plugging pose a challenge to normal airway clearance mechanisms. Patients who are intubated and mechanically ventilated share similar inadequacies in mobilizing and removing secretions. A weak, ineffective cough can be caused by physical restriction and the presence of an endotracheal tube, which inhibits the ability to clear secretions. Any breakdown in the normal airway clearance mechanism can result in secretion retention. Airway obstruction (partial or complete) may contribute to atelectasis and can result in inadequate ventilation and gas exchange.[6] The goal of airway clearance therapy is to promote improvement in cough and to facilitate expectoration by using techniques and modalities that can meet specific airway clearance objectives.

Historically, CPT has been the accepted standard for airway clearance therapy in pediatric patients and cystic fibrosis patients.[7] In recent years alternatives have become available and are often compared to conventional CPT for the amount of sputum produced and their ability to re-expand areas of atelectasis and improve gas exchange. In CPT, positioning, gravity drainage, and percussion and vibration are effective in moving secretions from the small to the large airways, allowing sputum expectoration by cough. CPT in combination with kinetic therapy has been shown to be effective in reducing atelectasis in critically ill patients.[8] Kinetic therapy used in combination with bronchodilators may parallel the results of IPV with kinetic therapy for the resolution of atelectasis. We did not use kinetic therapy on any patient included in the present studies.

Positive expiratory pressure, another modality that came from Europe, was designed to promote secretion clearance by active exhalation through a flow resister. The positive pressure created in the airway on exhalation assists in opening the small airways, allowing mobilization of secretions and cough. PEP was compared to CPT in multiple airway clearance modality evaluations of patients as young as 3 years, with positive results seen primarily in postoperative atelectasis patients and cystic fibrosis patients.[6] Oscillatory PEP devices such as the Flutter valve and the Acapella device are designed to vibrate the airway walls and thus promote mucus clearance while maintaining a degree of PEP to keep airways open during exhalation. The Flutter valve is gravity dependent, unlike the Acapella, which is not dependent on patient position and can be used with children, with a mask. Adult and pediatric patients can operate the Flutter or the Acapella independent of a caregiver.

HFCWC (with *The Vest*) is another modality that can be performed independently; it is an acceptable alternative to CPT and has been successfully used on mechanically ventilated patients.[9] IPV, a combination of PEP or Flutter and aerosol therapy, allows chest percussion with low tidal volume, promoting mobilization of secretions from small to large airways and also promoting cough. IPV via mouthpiece, mask, or artificial airway is in its early stages of development for pediatric patients.

Studies comparing IPV, HFCWC, and CPT revealed that IPV was as effective as traditional CPT combined with aerosol therapy. Langenderfer compared the alternative airway clearance modalities such as HFCWC, Flutter, PEP, and IPV to conventional CPT and concluded that IPV and HFCWC uniquely benefited patients who can't perform other therapies.[1] Reports of IPV for conditions other than cystic fibrosis found radiographic improvement in segmental atelectasis within 48 hours of initiating treatment.[4] Homnick et al presented a comparative trial of IPV versus CPT, involving 16 cystic fibrosis patients. They concluded that IPV was as effective as CPT combined with aerosol therapy in protecting lung function.[7]

The results from our retrospective study were similar to results from prior studies, including a clinically important improvement in atelectasis when using IPV therapy as the airway clearance modality. To better understand and validate the clinical effects of IPV, the randomized, controlled trial was conducted. Our clinical trial paralleled other trials by comparing IPV to CPT and postural drainage. Chest radiographs provided objective measurements for assessing changes in atelectasis score from baseline and helped guide and determine the duration of therapy. In addition we hypothesized that if atelectasis improved, lung volume would increase and therefore static compliance might be affected. The fact that compliance showed no change may be explained in a variety of ways, including the fact that simply increasing lung volume does not necessarily change the pressure-volume characteristics (the curve may simply be shifted upwards). Also, the effect on lung volume may have been too small to affect compliance.

Limitations of the randomized, controlled trial included relatively small sample size and a lack of control for severity of illness or any other aspect of care. However, in the CPT group, even excluding the patient who worsened following treatment, the results would have been the same because no patient showed improvement in atelectasis score. On the other hand, all but 1 IPV patient showed improvement following treatment. Though a Type I error could have been made in concluding that there was a difference in atelectasis scores, the probability of that event is $< 1$ in 1,000. Also, there was no difference between the initial atelectasis scores of the CPT and IPV groups (median 2.0 vs 2.0, $p = 0.530$), which leads us to believe that the 2 groups were comparable. The atelectasis score is a subjective evaluation, which could cause some inconsistency in results. However, the physicians responsible for assigning the scores were blinded to the treatment type, decreasing the chance of bias.

If a further study is undertaken based on our data, we would like to know the sample size required to show a difference in treatment effect of a given size while maintaining a statistical power of at least 0.80. Unfortunately there appears to be no power analysis procedure for comparing median values of ordinal data.[10,11] One alternative is to postulate that the CPT group and the IPV group have an equal probability of showing an improved atelectasis score after treatment (ie, the null hypothesis). We define a clinically important change in atelectasis score as being 1.0 unit. We further hypothesize that the probability of a score improving by at least 1.0 unit by chance is 0.50. We can now do a power analysis for the difference between 2 proportions, assuming 2 tails, with $\alpha$ set at 0.05 and power at 0.80. Our data suggest that 86% of patients treated with IPV will show an improvement of at least 1.0 unit. To detect that proportion compared to a control group in which 50% of the patients showed an improvement of 1.0 unit, we would need to enroll 25 patients in each group. If only 10% of the control group showed improvement, we would need to enroll only 6 patients in each group to get a power of 0.85. Given that none of the control patients showed any improvement in our study, the smaller sample size might be achievable.

## Conclusions

The results of this study suggest that IPV is a more effective method of re-expanding areas of atelectasis in ventilated patients than is conventional CPT. IPV may achieve results in about half the number of treatment days as CPT, with no adverse reactions. Given that both CPT and IPV treatments last 10–15 min, we speculate that IPV may be associated with a lower cost of care, through reduced labor hours.

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# Accuracy of Oxygen Analyzers at Subatmospheric Concentrations Used in Treatment of Hypoplastic Left Heart Syndrome

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**INTRODUCTION: The immediate survival of infants with hypoplastic left heart syndrome depends on success in achieving several therapeutic goals: (1) maintain patency of the ductus arteriosus, (2) assure adequate mixing of blood at the atrial level, and (3) establish and maintain a balance between systemic and pulmonary blood flow at or near unity. In accomplishing that final goal, various ventilatory strategies have been used to alter the physiologic modifiers of pulmonary vascular resistance and thus maintain balanced circulation, including ventilation with gas of subatmospheric oxygen concentration. However, no data on this subject have been published in the scientific literature, and commercial oxygen analyzers are specified for use within the range of 0.21 to 1.0 fraction of inspired oxygen ($F_{IO_2}$), leaving the accuracy of hypoxic gas delivery somewhat uncertain. We evaluated the performance of oxygen analyzers below $F_{IO_2}$ 0.21. METHODS: Two commercially available analyzers were studied: the TED-190 (Teledyne) and the Mini-OX III. Five new analyzers of each model were tested. After a 2-point calibration ($F_{IO_2}$ 1.0 and 0.21), all 5 analyzers of the same model were simultaneously exposed to precision-blended gases at 6 different concentrations of oxygen in nitrogen. Steady state was maintained for at least 2 min at each concentration before readings were recorded. Calibration was verified at $F_{IO_2}$ 0.21 between each level. RESULTS: The mean ± SD error was 0.0013 ± 0.0021 for the Mini-OX III analyzers and −0.0004 ± 0.0009 for the Teledyne analyzers. The upper and lower limits of the 95% confidence interval were 0.39% and −0.13% for the Mini-OX III analyzers and 0.07% and −0.15% for the Teledyne analyzers. The maximum difference between measured and known oxygen concentrations was 1% of full scale. CONCLUSIONS: The Mini-OX III and the Teledyne TED-190 provide accurate and reliable $F_{IO_2}$ readings between 0 and 0.21 that are within the manufacturers' specifications for maximum error. These 2 analyzers are therefore acceptable for use in delivering subambient oxygen concentrations. The Mini-OX III displays oxygen concentration to the nearest 0.1% and may be more appropriate for precise control.**

*Key words: oxygen analyzer, subatmospheric concentration, hypoplastic left heart syndrome.* [Respir Care 2002;47(10):1168–1172]

## Introduction

Hypoplastic left heart syndrome (HLHS) is a potentially fatal congenital heart defect that occurs with failure of the systemic or left heart structures to develop adequately. HLHS describes a spectrum of cardiac abnormalities characterized by marked hypoplasia of the left ventricle and ascending aorta.[1] HLHS affects 0.016–0.036% of live births and 1.4–3.8% of patients with congenital heart defect.[2] Congenital heart defect is more prevalent in males than females, with a male predominance of 60–70%.[3] Without surgical intervention, the majority of infants born with HLHS will die within a month of birth.

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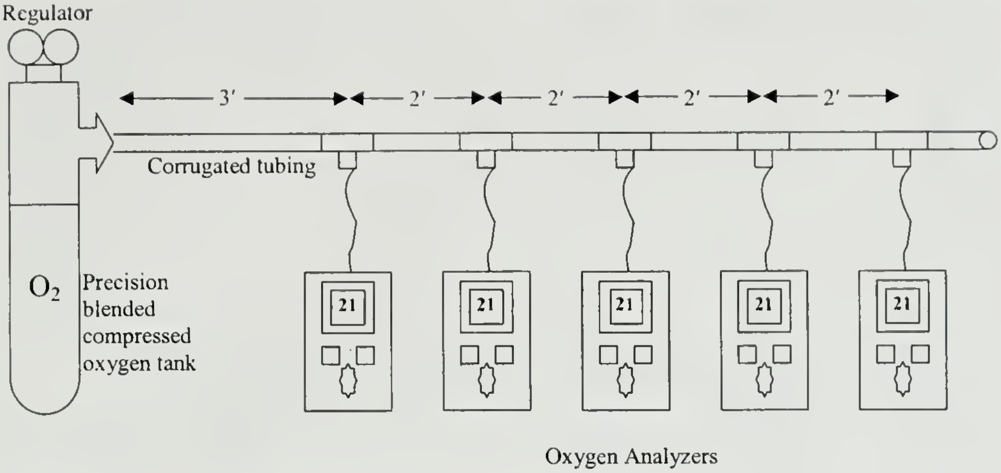

Fig. 1. Gas reservoir system for oxygen analyzer testing.

Infants born with HLHS have various degrees of hypoplasia or atresia of the aorta, aortic and mitral valves, and left ventricle. Typically, a patent foramen ovale or ductus arteriosus or a true atrial septal defect must be present to allow for left-to-right intracardiac shunting. Four distinct anatomical subtypes, based on the morphology of the left heart valves, have been described: (1) aortic and mitral stenosis, (2) aortic and mitral atresia, (3) aortic atresia and mitral stenosis, and (4) aortic stenosis and mitral atresia.[4]

Because of these complex congenital malformations, oxygen-saturated pulmonary venous blood returning to the left atrium cannot flow into the left ventricle. This intracardiac shunting results in pulmonary venous blood flowing across the atrial septum and mixing with desaturated, systemic venous blood in the right atrium. This desaturated, mixed blood gets pumped to both the pulmonary (via the branch pulmonary arteries) and systemic (via the ductus arteriosus and descending aorta) circulations in parallel by the right ventricle. Thus, blood flow into each circulation depends on the pulmonary or systemic vascular resistance.

Frequently, optimization of systemic oxygenation and perfusion is achieved with very little medical intervention other than intravenous administration of prostaglandins to maintain ductal patency. However, with clinical signs of poor perfusion, metabolic acidosis, or oliguria it becomes medically necessary to increase the pulmonary vascular resistance by active respiratory management.

In most neonatal intensive care units oxygen analyzers are routinely used with oxygen hoods and mechanical ventilators to measure concentrations of oxygen administered to acutely ill patients. Four types of oxygen analyzer are commonly available: polargraphic, galvanic cell, paramagnetic, and Wheatstone bridge.[5] We chose to bench test 2 polargraphic analyzers because of their response time, design for continuous use, and the availability of both high and low oxygen concentration alarms. Commercially available oxygen analyzers are specified for use within the range of 0.21 to 1.0 fraction of inspired oxygen ($F_{IO_2}$). Within that $F_{IO_2}$ range both manufacturers of the analyzers we studied list an accuracy of ± 2%. With the accuracy of the analyzer uncertain we were unsure of our ability to adequately control a subatmospheric oxygen delivery system. The purpose of this bench study was to evaluate the accuracy of 2 oxygen analyzers below the measurement range specified by the manufacturer.

## Methods

Two commercially available brands of analyzer were evaluated: the TED-190 (Teledyne Analytical Instruments, City of Industry, California) and the Mini-OX III (Mine Safety Appliances Company, Pittsburgh, Pennsylvania). We purchased 5 new analyzers of each model specifically for this bench test. Per manufacturers' specifications, all 10 analyzers had a 2-point calibration procedure performed at $F_{IO_2}$ of 1.0 and 0.21 prior to the start of the bench test. Room temperature on the day of the bench study was 67° F.

For the purpose of this study we developed a testing reservoir using a 12 ft length of disposable corrugated tubing (Allegiance Healthcare, McGaw Park, Illinois). The corrugated tubing was cut and T-pieces were inserted at the following interval lengths: 3, 5, 7, 9 and 11 ft (Fig. 1).

After the analyzers were calibrated, all 5 analyzers of the same model were simultaneously exposed to the same concentration of precision-blended oxygen in nitrogen. All gas mixtures were independently certified by the supplier (AGA Gas, Cleveland, Ohio) for accuracy (measured by paramagnetic analysis with an analytical tolerance of ± 2%). We used oxygen concentrations of 0, 5, 10, 12, 15, and 100%. Steady state gas supply (12–15 L/min) was maintained from a 16 psi, size D cylinder source for a

Table 1. Oxygen Concentrations Data from TED-190 Analyzers

| Analyzer | Actual Concentration 0.00 | | Actual Concentration 0.0511 | | Actual Concentration 0.1010 | | Actual Concentration 0.1210 | | Actual Concentration 0.1510 | | Actual Concentration 0.2100 | | Actual Concentration 1.0 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Measured | Difference | Measured | Difference | Measured | Difference | Measured | Difference | Measured | Difference | Measured | Difference | Calibration | Difference |
| 1 | 0.0000 | 0.0000 | 0.0500 | −0.0011 | 0.1000 | −0.0010 | 0.1200 | −0.0010 | 0.1500 | −0.0010 | 0.2100 | 0.0000 | 1.0 | 0.0 |
| 2 | 0.0000 | 0.0000 | 0.0500 | −0.0011 | 0.1000 | −0.0010 | 0.1200 | −0.0010 | 0.1500 | −0.0010 | 0.2100 | 0.0000 | 1.0 | 0.0 |
| 3 | 0.0000 | 0.0000 | 0.0500 | −0.0011 | 0.1000 | −0.0010 | 0.1200 | −0.0010 | 0.1500 | −0.0010 | 0.2100 | 0.0000 | 1.0 | 0.0 |
| 4 | 0.0000 | 0.0000 | 0.0600 | 0.0089 | 0.1000 | −0.0010 | 0.1200 | −0.0010 | 0.1500 | −0.0010 | 0.2100 | 0.0000 | 1.0 | 0.0 |
| 5 | 0.0000 | 0.0000 | 0.0500 | −0.0011 | 0.1000 | −0.0010 | 0.1200 | −0.0010 | 0.1500 | −0.0010 | 0.2100 | 0.0000 | 1.0 | 0.0 |
| Mean | 0.0000 | 0.0000 | 0.0520 | 0.0009 | 0.1000 | −0.0010 | 0.1200 | −0.0010 | 0.1500 | −0.0010 | 0.2100 | 0.0000 | 1.0000 | 0.0000 |
| SD | 0.0000 | 0.0000 | 0.0045 | 0.0045 | 0.0000 | 0.0000 | 0.0000 | 0.0000 | 0.0000 | 0.0000 | 0.0000 | 0.0000 | 0.0000 | 0.0000 |
| Total Error (confidence interval for mean difference at 95% level) | | | | | | | | | | | | | | |
| Upper limit | | 0.00 | | 0.64 | | −0.10 | | −0.10 | | −0.10 | | 0.00 | | 0.00 |
| Lower limit | | 0.00 | | −0.46 | | −0.10 | | −0.10 | | −0.10 | | 0.00 | | 0.00 |

Table 2. Oxygen Concentrations Data from Mini-OX III Analyzers

| Analyzer | Actual Concentration 0.00 | | Actual Concentration 0.0511 | | Actual Concentration 0.1010 | | Actual Concentration 0.1210 | | Actual Concentration 0.1510 | | Actual Concentration 0.2100 | | Actual Concentration 1.0 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Measured | Difference | Measured | Difference | Measured | Difference | Measured | Difference | Measured | Difference | Measured | Difference | Calibration | Difference |
| 1 | 0.0090 | 0.0090 | 0.0590 | 0.0079 | 0.1050 | 0.0040 | 0.1220 | 0.0010 | 0.1520 | 0.0010 | 0.2090 | 0.0010 | 1.0 | 0.0 |
| 2 | 0.0020 | 0.0020 | 0.0520 | 0.0009 | 0.1020 | 0.0010 | 0.1220 | 0.0010 | 0.1520 | 0.0010 | 0.2080 | −0.0020 | 1.0 | 0.0 |
| 3 | 0.0000 | 0.0000 | 0.0500 | −0.0011 | 0.1020 | 0.0010 | 0.1210 | 0.0000 | 0.1520 | 0.0010 | 0.2230 | 0.0130 | 1.0 | 0.0 |
| 4 | 0.0010 | 0.0010 | 0.0510 | −0.0001 | 0.1000 | −0.0010 | 0.1200 | −0.0010 | 0.1510 | 0.0000 | 0.2090 | −0.0010 | 1.0 | 0.0 |
| 5 | 0.0000 | 0.0000 | 0.0530 | 0.0019 | 0.1020 | 0.0010 | 0.1210 | 0.0000 | 0.1510 | 0.0000 | 0.2110 | 0.0010 | 1.0 | 0.0 |
| Mean | 0.0024 | 0.0024 | 0.0530 | 0.0019 | 0.1022 | 0.0012 | 0.1212 | 0.0002 | 0.1516 | 0.0006 | 0.2120 | 0.0020 | 1.0 | 0.0 |
| SD | 0.0038 | 0.0038 | 0.0035 | 0.0035 | 0.0018 | 0.0018 | 0.0008 | 0.0008 | 0.0005 | 0.0005 | 0.0062 | 0.0062 | 0.0 | 0.0 |
| Total Error (confidence interval for mean difference at 95% level) | | | | | | | | | | | | | | |
| Upper limit | | 0.71 | | 0.63 | | 0.34 | | 0.12 | | 0.13 | | 0.97 | | 0.00 |
| Lower limit | | −0.23 | | −0.25 | | −0.10 | | −0.08 | | −0.01 | | −0.57 | | 0.00 |

minimum of 2 min at each concentration before readings were recorded. After using a specific concentration of gas, the reservoir system was flushed for 5 min with a 55 psi gas source with an oxygen concentration of 21%. Following the flush of the reservoir system, a 1-point calibration (using 21% oxygen) of the analyzers was performed prior to testing with the next gas concentration. This procedure was repeated for both the Teledyne and Mini-OX analyzers. Note that we were not testing the gas but, rather, the hypothesis that the analyzer is accurate. The measurement error was calculated as known $F_{IO_2}$ minus measured $F_{IO_2}$. The mean ± SD error was calculated. The confidence interval for mean error at the 95% level was calculated for both brands of analyzer at each study $F_{IO_2}$.

## Results

The mean ± SD error was 0.0013 ± 0.0021 for the Mini-OX III analyzers and −0.0004 ± 0.0009 for the Teledyne analyzers. The upper and lower limits of the 95% confidence interval were 0.39% and −0.13% for the Mini-OX III analyzers, and 0.07% and −0.15% for the Teledyne analyzers.

The maximum difference between measured and known oxygen concentrations was 1%. Table 1 shows the results for the Teledyne analyzers. Table 2 shows the results for the Mini-OX III analyzers. Figure 2 shows the mean error (as percentage of full scale), with error bars representing the 95% confidence intervals. The dotted lines represent the manufacturers' total error specifications.

## Discussion

Stabilizing an HLHS infant requires an equal balance between systemic and pulmonary blood flows. To achieve and maintain that critical balance, artificial manipulation of the pulmonary vascular resistance and systemic vascular resistance is often needed. Pulmonary vascular resis-

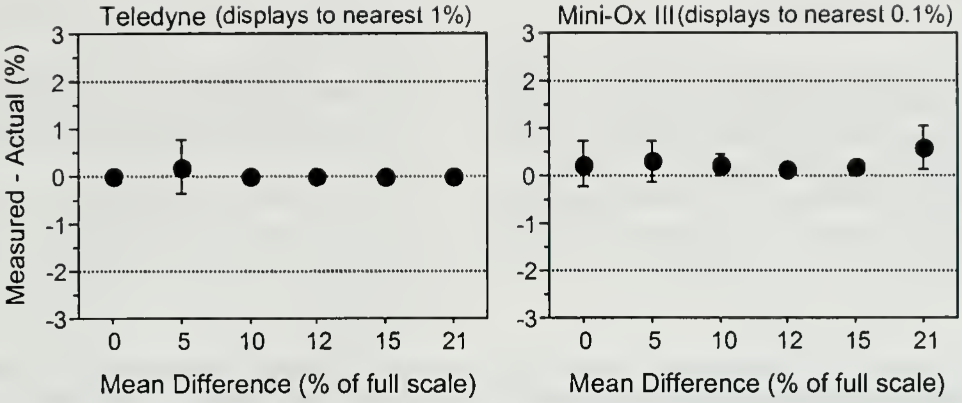

Fig. 2. Mean error (as percentage of full scale) of the measured versus actual oxygen concentrations. The error bars represent the 95% confidence intervals. The dotted lines represent the manufacturers' total error specifications.

tance can be manipulated by respiratory management in 2 ways.[2,6] Strategies to increase pulmonary vascular resistance involve titrating nitrogen or carbon dioxide during mechanical ventilation. Increasing $P_{aCO_2}$ to 45–50 mm Hg[7,8] or decreasing $P_{aO_2}$ to achieve blood oxygen saturation (measured via pulse oximetry) < 80%[9,10] can increase pulmonary vascular resistance. This can be accomplished by mechanical ventilation with hypoventilation or by the addition of nitrogen or carbon dioxide to the inspired gas.

Though the methodology of titrating carbon dioxide has been studied,[11,12] the methodology of delivering hypoxic gas mixtures (nitrogen bleed-in) has not been as closely scrutinized. Typically, $F_{IO_2}$ between 0.15 and 0.21 has been used to manipulate pulmonary vascular resistance in children with HLHS. A literature review of the use of hypoxic gas mixtures found very little scientific evidence on this subject other than a technical description of the delivery system. Delivering hypoxic gas mixtures to mechanically ventilated patients has been achieved by bleeding in low flows of nitrogen.

The methodology of nitrogen bleed-in often requires various flows to achieve the appropriate oxygen dilution, so hypoxic gas mixtures are ordered as $F_{IO_2}$ values instead of by nitrogen flow rates. In our clinical setting, $F_{IO_2}$ increases and decreases were frequently being ordered to exact concentrations. Though $F_{IO_2}$ in the desired range is easily obtained through nitrogen dilution, the ability of commercially available oxygen analyzers to accurately monitor those concentrations was unproven. Our study indicates that the manufacturers' specification of maximum error is easily maintained, down to 0% oxygen.

There are 2 potential limitations of the present study. The first is that we evaluated brand new analyzers of each model. All 10 of these analyzers had new fuel cells during this bench evaluation, and because these oxygen fuel cells deteriorate with age, the accuracy may decrease to below that reported in this study. Further study of aged oxygen fuel cells is necessary.

The second potential limitation is that these analyzers were tested at ambient atmospheric pressure (sea level). This verifies the analyzers for applications with a hood as the delivery system; it does not replicate the condition of elevated pressure during mechanical ventilation. However, assuming the mean airway pressure during mechanical ventilation is < 20 cm $H_2O$, that represents a deviation of < 2% from sea level pressure. Both polargraphic and galvanic cell analyzers measure $P_{O_2}$ and display percentage of oxygen. If the analyzers were calibrated while connected to the ventilator, we see no theoretical reasons that they should be any less accurate than our study indicates.

Both analyzers had less error than specified by the manufacturers. This is not surprising given that we were examining a small section of the instruments' range and thereby excluding nonlinearities that might be present over the entire range. Though an error specification of 2% of full scale is common among oxygen analyzers, it does not imply that they all will give results similar to those we studied, particularly if the signal from the oxygen sensor is digitized and linearized in any way before display.

An important clinical issue became evident during this bench study. Both analyzer brands tested had a default low-$F_{IO_2}$ alarm setting of 0.15. Thus, when we needed to use $F_{IO_2}$ of 0.15 we had to deal with a constant nuisance alarm. Because even minor $F_{IO_2}$ fluctuations can have a substantial effect on patient condition, the Mini-OX III, with readings to the nearest 0.1%, might allow for the most precise measurement of hypoxic gas mixtures. However, we selected the Teledyne analyzer for clinical use because the manufacturer modified the units so that the alarm thresholds could be adjusted down to 0%. This allowed us to set the low alarm threshold at 2% less than our delivered concentration for 0.15 and 0.16 without constant alarm.

## Conclusions

The Mini-OX III and the Teledyne TED-190 provide $F_{IO_2}$ readings between 0 and 0.21 that are well within the manufacturers' specifications for maximum error. These 2 analyzers are therefore acceptable for clinical use in measuring subambient concentrations of oxygen. The Mini-OX III displays oxygen concentration to the nearest 0.1% and may be more appropriate for precise control.

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# Battery Duration of Portable Ventilators: Effects of Control Variable, Positive End-Expiratory Pressure, and Inspired Oxygen Concentration

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**INTRODUCTION: Portable ventilators require battery power during transport or when alternating current is unavailable. Manufacturers report battery duration at nominal ventilator settings. METHODS: We studied the effects of control variable (pressure control vs volume control), positive end-expiratory pressure (PEEP), and fraction of inspired oxygen ($F_{IO_2}$) on the battery duration of 8 portable ventilators: Achieva, HT50, iVent201, LTV1000, TBird Advanced Ventilator System (AVS), Avian, Uni-Vent 750, and Uni-Vent 754. Each ventilator was set to ventilate a test lung at a rate of 10 breaths/min, tidal volume of 750 mL, and inspiratory time of 1.5 s, with volume-controlled ventilation and then pressure-controlled ventilation (PCV), if available. $F_{IO_2}$ was set at 0.21 and then 1.0. PEEP was set at 0, 10, and then 20 cm $H_2O$. Test lung compliance and resistance were set at 20 mL/cm $H_2O$ and 5 cm $H_2O$/L/s, respectively. Five trials were performed with each portable ventilator, with each combination of settings. Time to low-battery alarm, battery-empty alarm, and failure to ventilate the test lung were recorded. Portable ventilator performance during the trials was determined by continuous recording of tidal volume. RESULTS: The battery duration of pneumatically driven portable ventilators is longer than that of electrically driven portable ventilators. The battery duration of pneumatically driven portable ventilators is minimally affected by ventilator settings. The battery duration of electrically driven portable ventilators is shortened by use of PCV, increasing PEEP, and increasing $F_{IO_2}$. Compared to zero PEEP, PEEP of 20 cm $H_2O$ reduced battery duration with HT50 (40%), LTV1000 (37%), TBird AVS (34%), and Achieva (15%). Compared to volume-controlled ventilation, PCV reduced battery duration with the LTV1000 (48%) and TBird AVS (18%). Compared to $F_{IO_2}$ of 1.0, $F_{IO_2}$ of 0.21 reduced battery duration with the Uni-Vent 754 (37%). Compared to $F_{IO_2}$ of 0.21, $F_{IO_2}$ of 1.0 reduced battery duration with the LTV1000 (17%) and TBird AVS (15%). The iVent201 was unable to deliver the set tidal volume with PCV and 20 cm $H_2O$ PEEP. Low-battery alarms functioned properly on all the ventilators. CONCLUSIONS: Battery duration differs greatly among the portable ventilators tested. Clinicians must be aware that portable ventilator battery duration is affected by control settings, lung impedance characteristics, and portable ventilator characteristics. Battery duration may be shorter than that reported in the operator's manual for each portable ventilator tested.** *Key words: portable ventilators, mechanical respiration, transportation of patients, power sources, positive pressure ventilation.* [Respir Care 2002;47(10):1173–1183]

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Table 1. Physical Characteristics of the Portable Ventilators Tested

| Ventilator | Input Power | Driving Mechanism | Dimensions (cm) | Weight (kg) | Listed Battery Duration (h) | Battery Type |
|---|---|---|---|---|---|---|
| Achieva | Electric | Piston | 27.3 × 33.8 × 39.6 | 14.5 | 4*<br>1† | Gel cell, sealed lead acid |
| Avian | Electric and pneumatic | Compressed gas | 25.4 × 30.5 × 12.7 | 5.0 | Minimum of 11 | Sealed lead acid |
| HT50 | Electric | Piston pump | 26 × 27 × 20 | 7.5 | Up to 10‡ | Sealed lead acid |
| iVent201 | Electric | Variable speed turbine | 35 × 24 × 29 | 11 | 1–2§ | Sealed lead acid |
| LTV1000 | Electric | Constant speed turbine | 7.62 × 25.4 × 30.5 | 5.72 | Minimum 1‖ | Gel cell, sealed lead acid |
| TBird AVS | Electric | Variable speed turbine | 33.02 × 36.83 × 27.94 | 15.0 | 0.82¶<br>0.1** | Sealed lead acid |
| Uni-Vent 750 | Electric and pneumatic | Compressed gas | 29.2 × 11.4 × 22.9 | 4.5 | 9 | Sealed lead acid |
| Uni-Vent 754 | Electric and pneumatic | Electric diaphragm pump or compressed gas | 22.55 × 29.21 × 11.43 | 5.8 | Up to 3††<br>Up to 12‡‡ | Sealed lead acid |

*At nominal load: alternating current electricity, respiratory rate (f) = 10 breaths/min, tidal volume ($V_T$) = 1000 mL, fraction of inspired oxygen ($F_{IO_2}$) = 0.21, inspiratory time ($T_I$) = 1.5 s, positive end-expiratory pressure (PEEP) = 0 cm $H_2O$, inspiratory pressure = 30 cm $H_2O$

†At heavy load: alternating current electricity, f = 20 breaths/min, $V_T$ = 1,500 mL, $F_{IO_2}$ = 1.0, $T_I$ = 1.0 s, PEEP = 20 cm $H_2O$, inspiratory pressure = 60 cm $H_2O$

‡At nominal load, alternating current electricity, volume-controlled ventilation, f = 20 breaths/min, $V_T$ = 500 mL, $T_I$ = 1.0 s, PEEP = 0 cm $H_2O$, power-save = on

§Depending on inspiratory settings and load conditions

‖At nominal load: alternating current electricity, f = 15 breaths/min, $V_T$ = 800 mL, $F_{IO_2}$ = 0.21, $T_I$ = 1.5 s, PEEP = 5 cm $H_2O$, compliance = 50 mL/cm $H_2O$, resistance = 5.87 cm $H_2O$/L/s, battery temp = 25°C

¶At nominal load: alternating current electricity, f = 15 breaths/min, $V_T$ = 800 mL, peak flow = 60 L/min, PEEP = 5 cm $H_2O$, compliance = 50 mL/cm $H_2O$, resistance = 3.0 cm $H_2O$/L/s

**At heavy load: alternating current electricity, f = 30 breaths/min, $V_T$ = 1,500 mL, peak flow = 140 L/min, PEEP = 30 cm $H_2O$, compliance = 17.5 mL/cm $H_2O$, resistance = 4.0 cm $H_2O$/L/s

††Using internal compressor

‡‡Using external compressed gas

(Adapted from the ventilators' operator manuals.)

## Introduction

Portable ventilators require an internal power source (battery) for operation when alternating current (AC) power is unavailable. For home-care ventilation, battery power is essential for patient mobility and the resulting improved quality of life. During patient transport, battery power is essential for safe patient movement. Power for and control of ventilator operation can be accomplished by the gas source (pneumatic), a battery (electric), or both. Classification of portable ventilators requires definition of input power (electric, pneumatic, or both) and driving system (pneumatic, piston, turbine, or fluidics).[1,2] Each type of control and driving system has advantages and disadvantages depending on the application (home, hospital, transport).[3,4] Most new-generation portable ventilators are microprocessor-controlled and possess internal drive systems, both of which require a continuous source of electricity.

Pneumatically powered portable ventilators require a high-pressure (30–60 psi) gas source from either an external compressor or a compressed gas cylinder. Electrically powered portable ventilators generate gas pressure and flow by a piston, turbine, or internal compressor. The duration of operation of any portable ventilator depends on the power source (pneumatic and/or electric), driving system (pneumatic and/or electric), relative load (impedance to gas flow and minute ventilation requirement), capacity and type of battery used, and ventilator settings. Pneumatically powered ventilators have relatively high gas consumption and low electricity consumption, so their duration of operation may be affected more by the availability of compressed gas than by battery duration. Electrically powered and controlled ventilators may have lower gas consumption but usually require more electricity, so their duration of operation depends on battery duration.

Typically, manufacturers of portable ventilators report the battery duration at nominal ventilator settings and load (compliance and resistance) conditions. We evaluated the effects of control variable (volume-controlled ventilation [VCV] vs pressure-controlled ventilation [PCV]), positive end-expiratory pressure (PEEP), and fraction of inspired oxygen ($F_{IO_2}$) on the battery duration of 8 commercially available portable ventilators.

Table 2. Battery Duration of Portable Ventilators

| Settings | | | Battery Duration (min ± SD) | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Mode | PEEP | $F_{IO_2}$ | Achieva | Avian | HT50 | iVent201 | LTV1000 | TBird AVS | Uni-Vent 750 | Uni-Vent 754 |
| PCV | 0 | 0.21 | 143 ± 7 | NA | 642 ± 72 | 54.5 ± 5.7 | 58.6 ± 6.6 | 28.2 ± 0.8 | NA | NA |
| PCV | 10 | 0.21 | 118 ± 9 | NA | 583 ± 3 | 44.5 ± 5.6 | 52.9 ± 5.8 | 21 ± 1.6 | NA | NA |
| PCV | 20 | 0.21 | 108 ± 5 | NA | 421 ± 53 | * | 40.4 ± 6.1 | 16.3 ± 0.6 | NA | NA |
| PCV | 0 | 1.0 | 135 ± 3 | NA | NA | 57.3 ± 1.3 | 55.2 ± 9.9 | 24.3 ± 3.2 | NA | NA |
| PCV | 10 | 1.0 | 118 ± 2 | NA | NA | 39.7 ± 6.7 | 45.3 ± 11 | 19 ± 0.2 | NA | NA |
| PCV | 20 | 1.0 | 109 ± 2 | NA | NA | * | 37.5 ± 4.5 | 13.7 ± 1.2 | NA | NA |
| VCV | 0 | 0.21 | 153 ± 4 | NA | 631 ± 38 | 57 ± 5.4 | 114 ± 5.9 | 33.6 ± 4.1 | NA | 290.7 ± 1.5 |
| VCV | 10 | 0.21 | 144 ± 10 | NA | 521 ± 44 | 55 ± 9.9 | 92.5 ± 2.6 | 25.7 ± 1.2 | NA | 265.5 ± 4.0 |
| VCV | 20 | 0.21 | 138 ± 7 | NA | 388 ± 4 | 50 ± 8.5 | 72 ± 3.2 | 19.5 ± 1.7 | NA | 257.4 ± 6.3 |
| VCV | 0 | 1.0 | 149 ± 5 | 893 ± 52 | NA | 55 ± 9.9 | 93.7 ± 9.1 | 29.3 ± 2.1 | 897 ± 39 | 464.8 ± 15.6 |
| VCV | 10 | 1.0 | 141 ± 7 | 835 ± 41 | NA | 53 ± 1.4 | 72.7 ± 4.2 | 24 ± 1.7 | 902 ± 32 | 453.7 ± 12.0 |
| VCV | 20 | 1.0 | 125 ± 12 | 799 ± 42 | NA | 49.7 ± 4.9 | 61.4 ± 1.1 | 17.2 ± 4.4 | 895 ± 35 | 435.5 ± 25.4 |

PEEP = positive end-expiratory pressure

$F_{IO_2}$ = fraction of inspired oxygen

PCV = pressure-controlled ventilation

VCV = volume-controlled ventilation

NA = not applicable: ventilator not tested at this condition because of unavailability of PCV or integral air-oxygen blender

* = ventilator not able to adequately ventilate the test lung at this condition

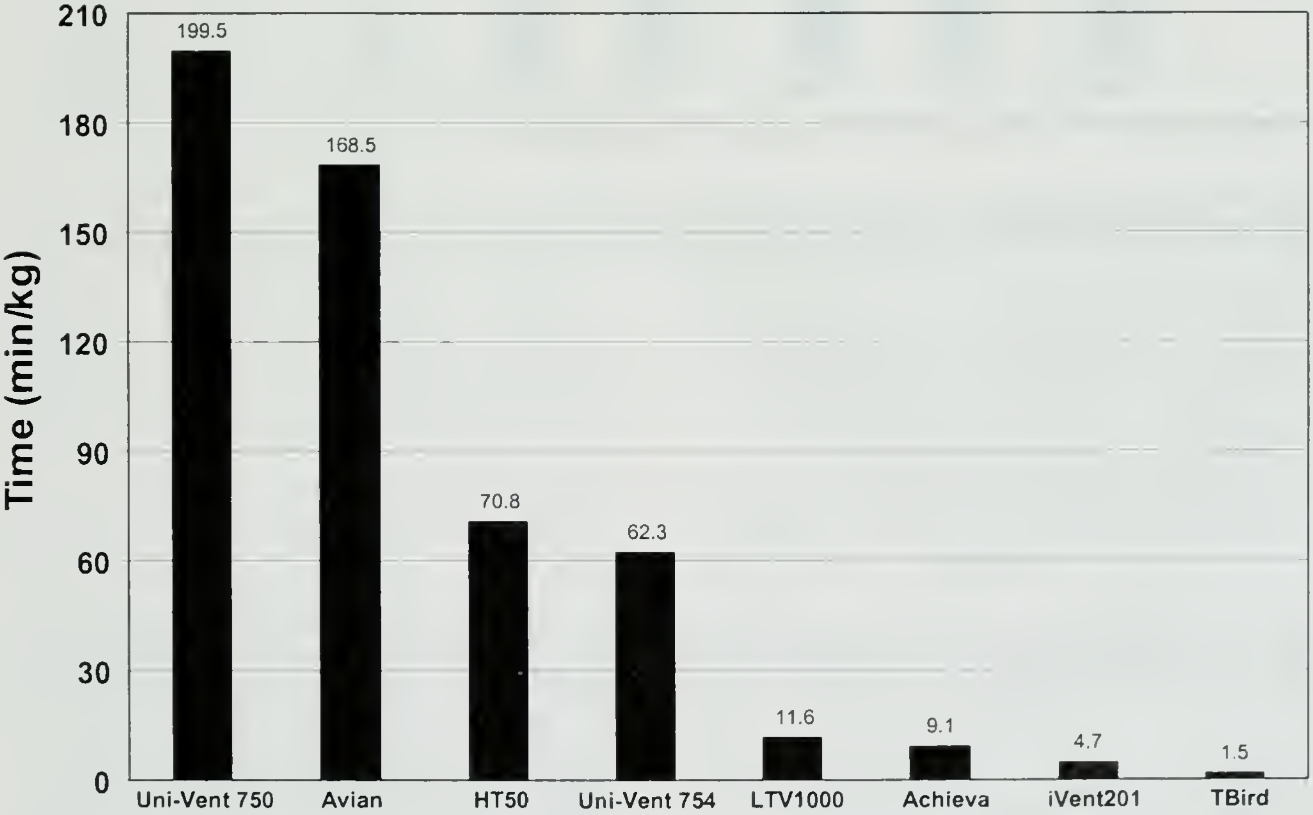

Fig. 1. Battery duration (mean of all test conditions) relative to the weight of each ventilator tested.

## Methods

We studied 8 electrically controlled portable ventilators with internal batteries: Achieva (Mallinckrodt/Puritan-Bennett, Carlsbad, California); HT50 (Newport Medical Instruments, Newport Beach, California); iVent201 (VersaMed, Rochelle Park, New Jersey); Advanced Ventilator System (AVS) LTV1000 (Pulmonetic Systems, Colton,

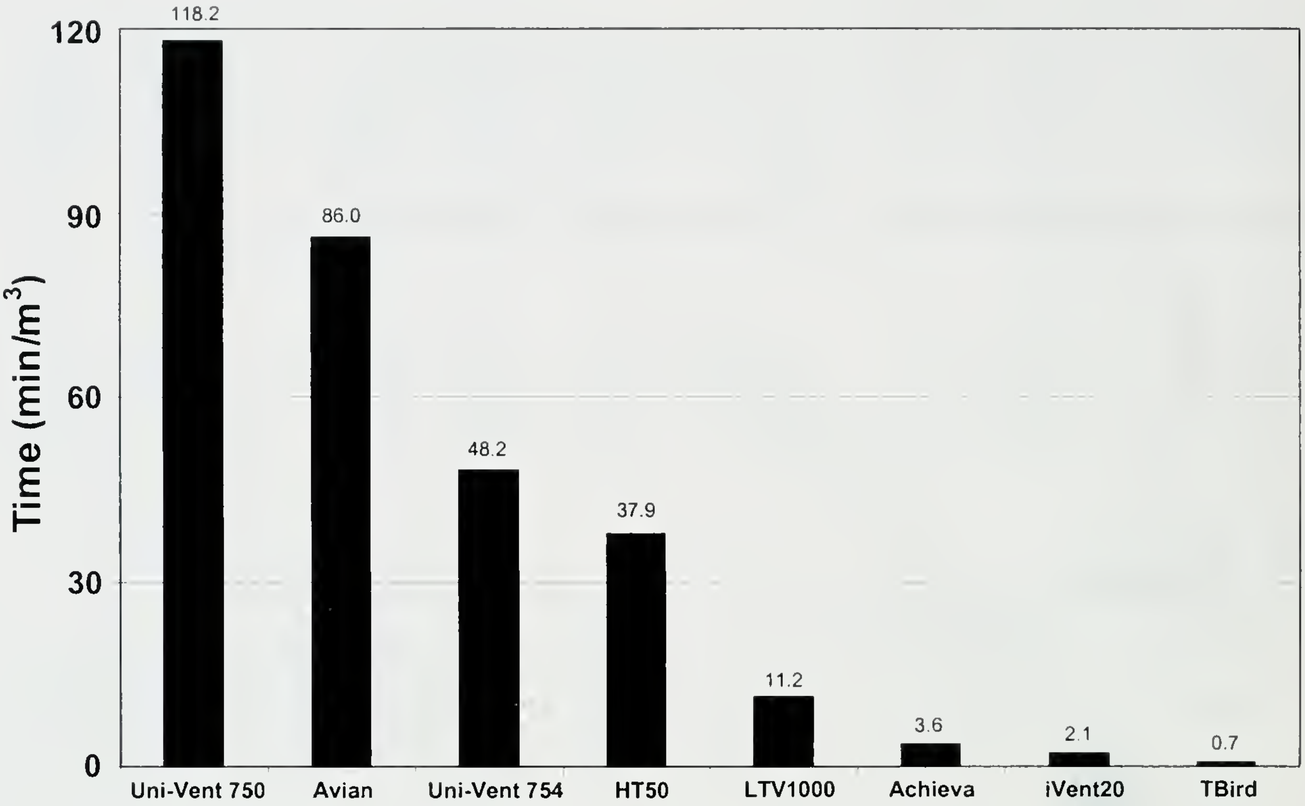

Fig. 2. Battery duration (mean of all test conditions) relative to the size of each ventilator tested.

Table 3. Battery Duration Difference with Increase in Positive End-Expiratory Pressure

| PEEP Change | Mean Change in Battery Duration (min)* with Increase in PEEP | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Achieva | Avian | HT50 | iVent201 | LTV1000 | TBird AVS | Uni-Vent 750 | Uni-Vent 754 |
| 0–10 cm $H_2O$ | −14.7 (−10.1%) | −58 (−6.5%) | −84.5 (−13.3%) | −7.9 (−14.1%) | −14.5 (−18.1%) | −6.4 (−22.3%) | +4.4 (+0.5%) | −18.2 (−4.8%) |
| 10–20 cm $H_2O$ | −10.5 (−8.0%) | −35.7 (−4.3%) | −147.1 (−26.7%) | +1.8 (+3.8%)† | −13.0 (−19.8%) | −5.8 (−25.6%) | −6.7 (−0.7%) | −13.2 (−3.7%) |
| 0–20 cm $H_2O$ | −25.2 (−17.3%) | −93.7 (−10.5%) | −231.6 (−36.4%) | −6.1 (−10.9%)† | −27.6 (−34.3%) | −12.2 (−42.2%) | −2.3 (−0.3%) | −31.3 (−8.3%) |

PEEP = positive end-expiratory pressure

*Values are the means of all conditions using each PEEP setting for each ventilator

†Does not include data from conditions of pressure-controlled ventilation with 20 cm $H_2O$ PEEP

California); TBird Advanced Ventilator System (AVS) and Avian (VIASYS Healthcare/Bird Products, Palm Springs, California); and Uni-Vent 750 and Uni-Vent 754 (Impact Instrumentation, West Caldwell, New Jersey). No external batteries were used during the evaluation. Table 1 lists the ventilators' input power, driving source, dimensions, weight, battery type, and battery duration, from each ventilator's operator manual.

Each ventilator was set to deliver a tidal volume ($V_T$) of 750 mL at a rate of 10 breaths/min and an inspiratory time of 1.5 s, to 1 side of a 2-chamber test lung (Model 1600 TTL, Michigan Instruments, Ann Arbor, Michigan) set to a compliance of 0.02 L/cm $H_2O$ and a resistance of 5 cm $H_2O$/L/s. Each ventilator was tested using VCV. The ventilators capable of PCV were also evaluated using PCV, with the inspiratory pressure adjusted to maintain a measured exhaled $V_T$ of 750 mL. During PCV, inspiratory time, respiratory frequency, and all other variables were held constant. Pneumatically driven ventilators without an internal air-oxygen blender were only tested at $F_{IO_2}$ of 1.0. Electrically driven ventilators that have an internal blender were evaluated at $F_{IO_2}$ of 0.21 and then 1.0. Electrically driven ventilators that do not have an internal blender were only evaluated at $F_{IO_2}$ of 0.21. Each ventilator was evaluated at PEEP levels of 0, 10, and then 20 cm $H_2O$. On ventilators that do not provide integral PEEP control, PEEP

Table 4. Battery Duration Difference Between Pressure-Controlled and Volume-Controlled Ventilation

| | Achieva | HT50 | iVent201 | LTV1000 | TBird AVS |
|---|---|---|---|---|---|
| Mean difference in battery duration (time with PCV − time with VCV) (min) | −19.8 (−14.0%) | +35.1 (+6.8%) | −4.3 (−8.0%)* | −36.1 (−42.7%) | −4.5 (−18.0%) |

*Does not include data from conditions of pressure-controlled ventilation with 20 cm $H_2O$ PEEP
PCV = pressure-controlled ventilation
VCV = volume-controlled ventilation

Table 5. Battery Duration Difference Between $F_{IO_2}$ of 1.0 and $F_{IO_2}$ of 0.21

| | Achieva | iVent201 | LTV1000 | TBird AVS | Uni-Vent 754 |
|---|---|---|---|---|---|
| Mean difference in battery duration (time with $F_{IO_2}$ 1.0 − time with $F_{IO_2}$ 0.21) (min) | −4.5 (−3.3%) | −1.3 (−2.4%) | −10.8 (−15.0%) | −2.8 (−11.6%) | +180.1 (+66.4%) |

$F_{IO_2}$ = fraction of inspired oxygen

was provided with an adjustable PEEP valve (Mercury Medical, Clearwater, Florida) placed at the exhalation valve. PEEP level was confirmed by measuring proximal airway pressure with a calibration analyzer (RT200, Allied Healthcare Products, St Louis, Missouri).

Each ventilator tested was up to date with respect to preventive maintenance schedule and passed all required pre-use calibration/verification procedures recommended by each manufacturer. Testing with the Achieva, HT50, Uni-Vent 754, and TBird AVS was accomplished with the same ventilator, based on availability. Testing with the Avian, Uni-Vent 750, iVent201, and LTV1000 were done with 2 of each ventilator model, and each ventilator was used at least once for each test condition.

Each ventilator battery was charged per the manufacturer's recommendation prior to testing. Each ventilator was attached to the test lung and operated on AC power for 5 min, during which all ventilator settings were set and confirmed. Each test run was initiated by disconnecting the AC power cord from the ventilator. Notation was made when any indication (visual or audible alarm) was made to signal loss of AC power. Each ventilator was monitored continuously until inspiratory flow output ceased. Time elapsed from initiation of test to the first low-power alarm, subsequent alarms, and ventilator failure were recorded. A minimum of 5 test runs were performed with each ventilator at each combination of test conditions.

We also evaluated the ability of each portable ventilator to deliver the set $V_T$ as battery power diminished. During the test with VCV and PEEP of 20 cm $H_2O$, exhaled $V_T$ was continuously measured by a pneumotachograph (RSS 100, Hans Rudolph, Kansas City, Missouri) between the test lung and breathing circuit. The electrically driven ventilators were tested at $F_{IO_2}$ of 0.21. Pneumatically driven portable ventilators were tested at $F_{IO_2}$ of 1.0.

Statistical analysis included analysis of variance for the effect of PEEP and Student's *t* test for the effect of control variable and $F_{IO_2}$, when available. Differences were considered statistically significant when p was $< 0.01$.

## Results

### General

Only 3 of the ventilators (Achieva, LTV1000, and TBird AVS) could be tested at all test conditions. The iVent201 was tested at all conditions except the 2 conditions that included the combination of PCV and PEEP of 20 cm $H_2O$, under which it was not capable of delivering the target $V_T$. The iVent201 alarmed and defaulted to an "open loop" mode that would allow spontaneous breathing within 10 min at that combination of settings. The HT50 was the only ventilator not tested with an $F_{IO_2}$ of 1.0, because it does not have an internal blender (a pneumatic oxygen accumulator is available). The Uni-Vent 754 was tested at all conditions except those including PCV, which is not available with the Uni-Vent 754. The Uni-Vent 750 and Avian were only tested at $F_{IO_2}$ of 1.0 with VCV because these ventilators lack an internal blender, are pneumatically powered, and do not offer traditional PCV.

### Total Battery Duration

Table 2 shows the total battery duration of each ventilator at all conditions available for evaluation. All the pneumatically driven ventilators exceeded 7 hours of total operation on battery, with the Uni-Vent 750 lasting nearly 15 hours. Mean battery duration of the electrically driven ventilators ranged from < 30 min (TBird AVS) to > 8 hours (HT50).

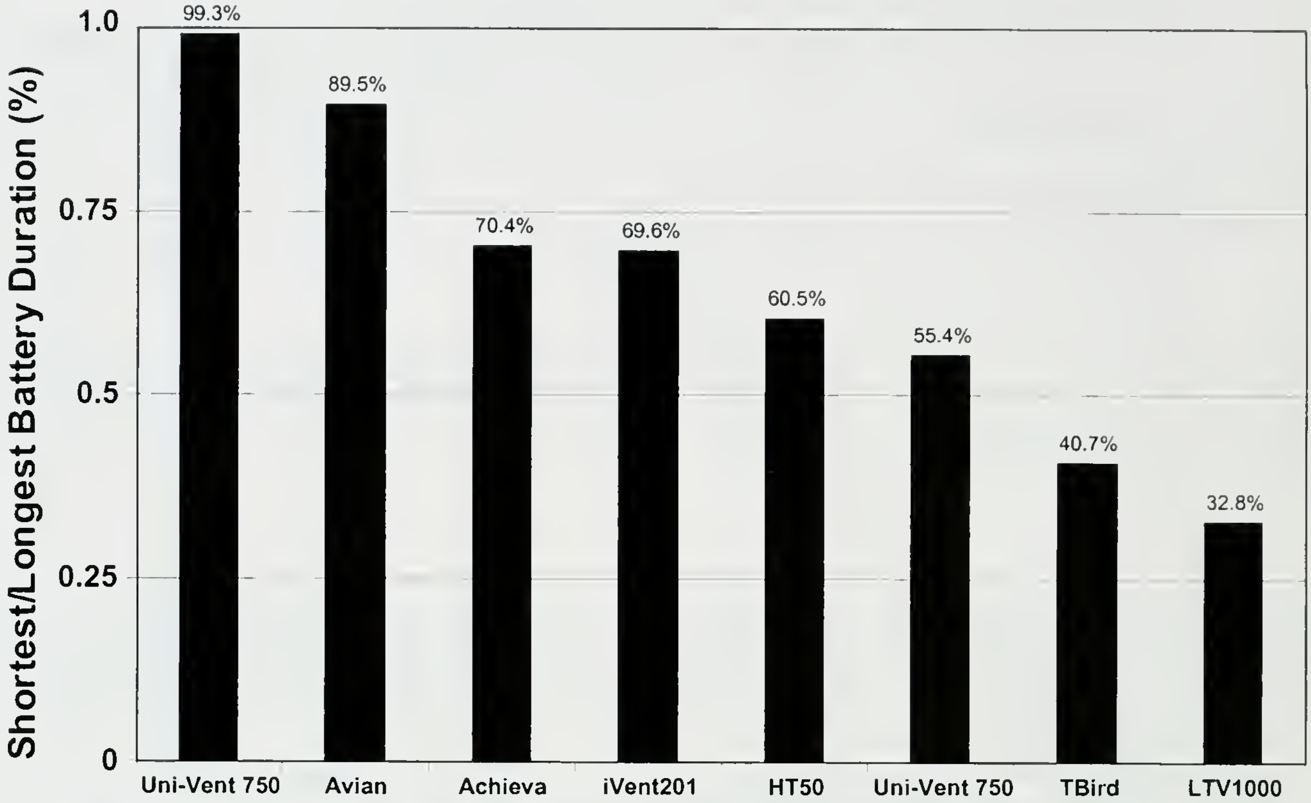

Fig. 3. Greatest change in battery duration as a result of ventilator settings (worse-case scenario) for each ventilator tested. The values represent the percent change (shortest duration/longest duration), and 100% equals the longest measured duration condition.

Figure 1 shows the mean battery durations relative to ventilator weight. Figure 2 shows the mean battery durations relative to ventilator dimensions.

### Effect of Ventilator Settings on Battery Duration

**Effect of PEEP.** Table 3 shows the mean reduction in battery duration resulting from incremental PEEP increases. The battery duration of the pneumatically driven ventilators was less affected by the addition of PEEP than the electrically driven ventilators. The Uni-Vent 750 was the only ventilator unaffected by the addition of PEEP. When used as a pneumatically driven device (with $F_{IO_2}$ 1.0) the battery duration of the Uni-Vent 754 was reduced by 2.4% with the addition of 10 cm $H_2O$ PEEP and by 6.3% with the addition of 20 cm $H_2O$ PEEP. When used as an electrically driven device (with $F_{IO_2}$ 0.21) the battery duration of the Uni-Vent 754 was reduced by 8.7% with the addition of 10 cm $H_2O$ PEEP and by 11.1% with the addition of 20 cm $H_2O$ PEEP. The battery duration of the TBird AVS was affected most by the addition of PEEP; battery duration was reduced by 22% and 42% with the addition of 10 and 20 cm $H_2O$ PEEP, respectively. The effect of PEEP on battery duration of the iVent201 is skewed because this ventilator could not complete the testing at the conditions combining PCV and 20 cm $H_2O$ PEEP.

**Effect of Control Variable (PCV vs VCV).** Three of the ventilators (Uni-Vent 750, Uni-Vent 754, and Avian) do not offer PCV and were therefore excluded from this portion of the testing. Table 4 shows the mean reduction in battery duration resulting from changing the control variable from VCV to PCV. The battery duration of the HT50 was nearly 7% longer with PCV than with VCV. The battery duration of all the other ventilators was shorter with PCV. The battery duration of the LTV1000 was most affected by the use of PCV; battery duration was reduced by nearly 43%. The effect of control variable on battery duration of the iVent201 may be underestimated because the condition of PCV with 20 cm $H_2O$ PEEP could not be performed.

**Effect of $F_{IO_2}$ Setting.** Three of the ventilators (Avian, HT50, and Uni-Vent 750) do not have an internal blender and were therefore excluded from this portion of the testing. Table 5 shows the mean reduction in battery duration from changing the $F_{IO_2}$. The Uni-Vent 754 was most af-

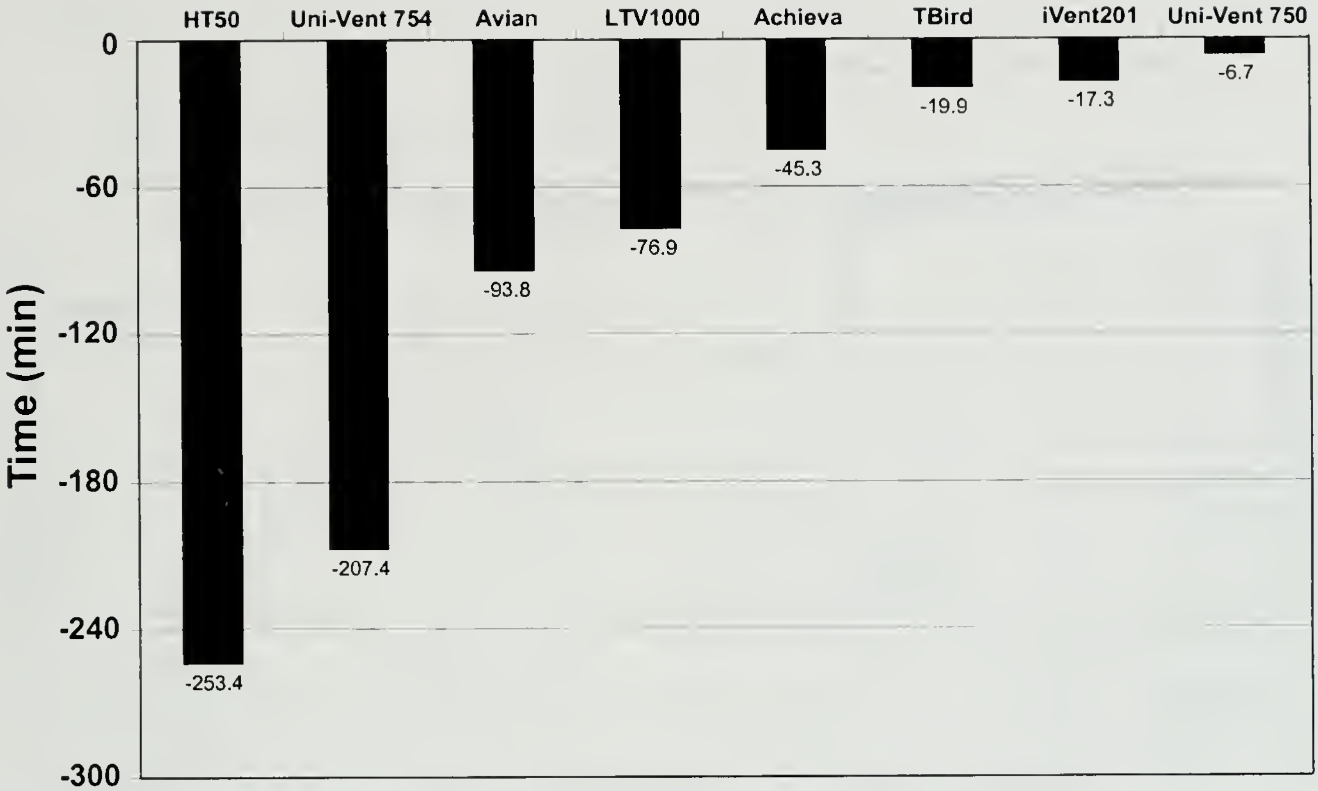

Fig. 4. Greatest change in battery duration as a result of ventilator settings for each ventilator tested. The values represent the time reduction in battery duration resulting from the worse-case scenario, calculated by the following formula: time change = time (condition with shortest duration) – time (condition with longest duration).

Table 6. Timing of Alarms*

| | Achieva | Avian | HT50 | iVent201 | LTV1000 | TBird AVS | Uni-Vent 750 | Uni-Vent 754 |
|---|---|---|---|---|---|---|---|---|
| Mean operation time on battery when first low-battery alarm sounds (min) | 27.7 (20.9%) | 101.3 (12.1%) | 196.0 (36.4%) | 18.0 (34.8%) | 23.0 (36.6%) | 13.7 (61.6%) | 150.7 (16.8%) | 53.0 (14.3%) |
| Mean operation time on battery when final battery-empty alarm sounds (min) | 20.4 (15.4%) | NA | 40.8 (7.6%) | 12.1 (23.2%) | 9.7 (16.1%) | 7.3 (32.5%) | NA | NA |

NA = Not applicable, battery-empty alarm not available on this ventilator
*Time = min (%) of battery life remaining at time of alarm

fected by $F_{IO_2}$ setting; battery duration was 66% longer with $F_{IO_2}$ of 1.0 than with $F_{IO_2}$ of 0.21. The battery duration of the other 4 ventilators was shorter with $F_{IO_2}$ of 1.0 than with $F_{IO_2}$ of 0.21.

**Effect of Combined Settings Changes.** Figure 3 shows the worst-case reduction in battery duration for each ventilator, as a percentage of total battery duration, based on the combination of settings tested, using the following formula:

Worst-case battery reduction =

shortest battery duration/longest battery duration

Figure 4 shows the worst-case reduction in battery duration in terms of time. Overall, the effect of ventilator settings on battery duration was not pronounced with the pneumatically driven ventilators: the worst-case reduction was 10.5% with the Avian and 0.7% with the Uni-Vent

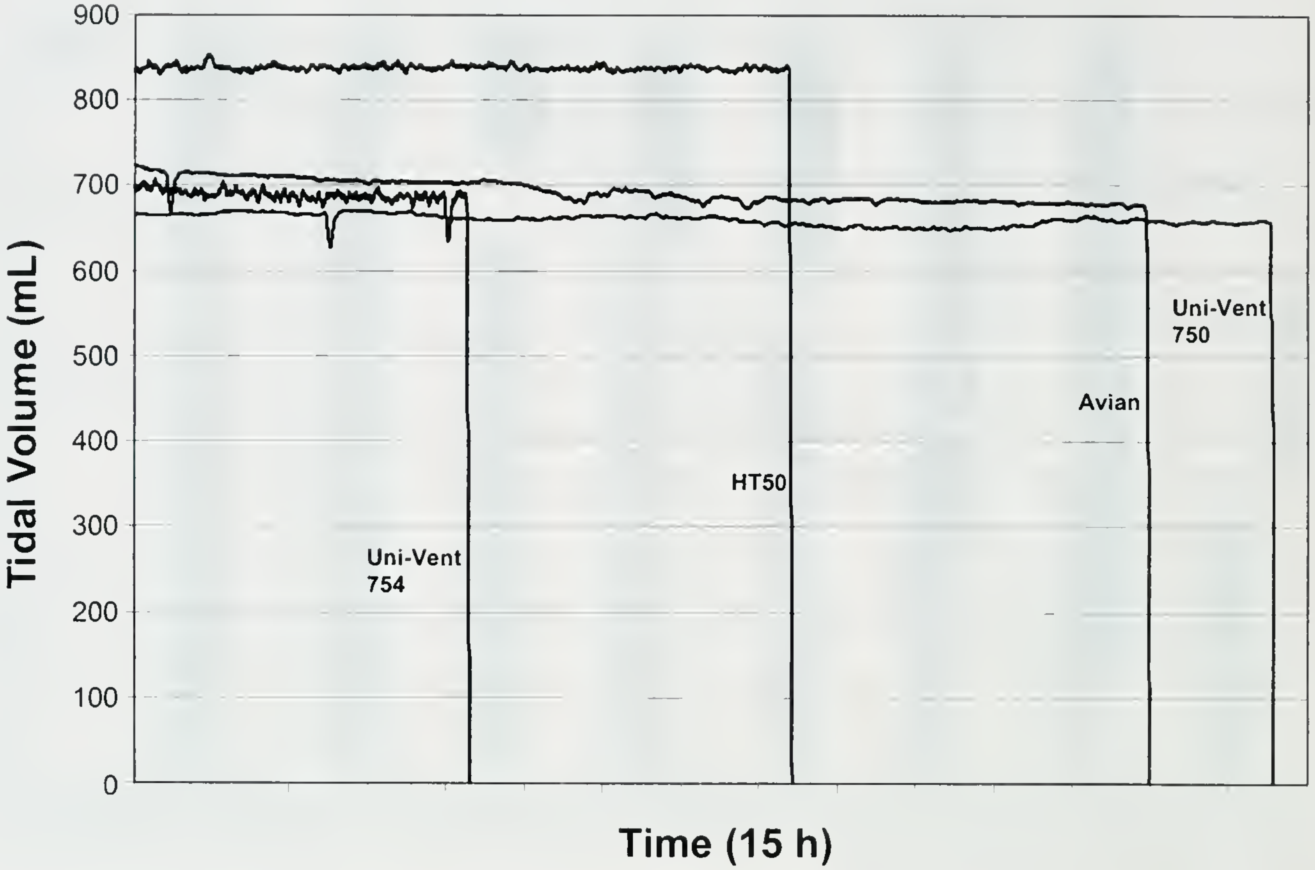

Fig. 5. Measured exhaled tidal volume during the terminal battery testing for portable ventilators that operated > 3 hours on battery power. The test conditions were: volume-controlled ventilation with positive end-expiratory pressure of 20 cm $H_2O$ for all ventilators; fraction of inspired oxygen ($F_{IO_2}$) 1.0 with the Uni-Vent 750 and Avian; $F_{IO_2}$ 0.21 with the Uni-Vent 754 and HT50.

750. The Uni-Vent 754 must be considered separately, as it performs as a pneumatically driven ventilator when set to deliver $F_{IO_2}$ of 1.0 and behaves as an electrically driven device when set to $F_{IO_2}$ of 0.21. With the electrically driven ventilators there were dramatic changes in battery duration resulting from ventilator setting changes. Worst-case reduction ranged from 29.6% with the Achieva to 77.3% with the LTV1000. Typically, the worst case (shortest battery duration) was associated with the use of PCV, $F_{IO_2}$ of 1.0, and 20 cm $H_2O$ PEEP, and the best case (longest battery duration) was associated with VCV, $F_{IO_2}$ of 0.21, and zero PEEP.

### Alarm Function

Each electrically driven ventilator gave an audible and visual alarm to signal loss of AC power. The pneumatically driven ventilators (Uni-Vent 750 and Avian) gave only a visual indication that AC power was lost. Table 6 shows the mean battery duration when the first low-battery alarm sounded and the mean battery duration when the final battery-empty alarm sounded (if available). The TBird AVS had the shortest time to low-battery alarm, in terms of both time (10.3 min) and percentage of total battery duration (44% depleted). The Avian had the longest time to low-battery alarm in terms of both time (744.7 min) and percentage of total battery duration (88.4% depleted). The TBird AVS gave the shortest warning time between final battery-empty alarm and ventilator failure (6.7 min). The Avian gave the final battery-empty alarm with the lowest percentage of remaining battery power (95.2% depleted, which was associated with 40 min of battery power remaining). The Uni-Vent 750 gave the longest warning time of impending ventilator failure (186 min).

### Tidal Volume Delivery As Battery Power Diminishes

Figure 5 shows the measured exhaled $V_T$ throughout the terminal battery testing for portable ventilators that lasted > 3 hours during VCV with 20 cm $H_2O$ PEEP. Figure 6 shows the measured exhaled $V_T$ throughout the terminal battery testing for portable ventilators that lasted < 3 hours during VCV with 20 cm $H_2O$ PEEP. The iVent201 was the only portable ventilator unable to maintain a constant $V_T$

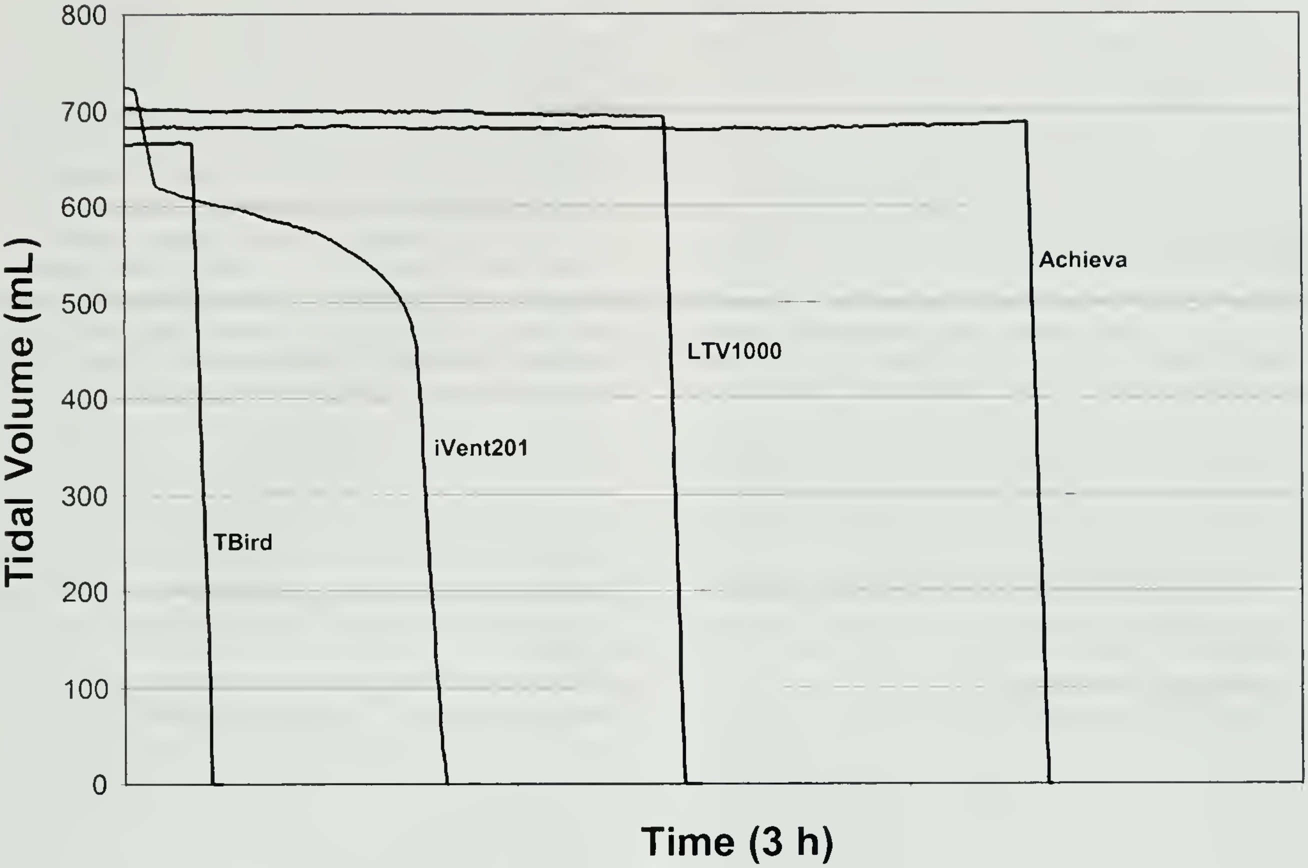

Fig. 6. Measured exhaled tidal volume during the terminal battery testing for portable ventilators that operated for < 3 hours on battery power. The test conditions were: volume-controlled ventilation, positive end-expiratory pressure of 20 cm $H_2O$, and fraction of inspired oxygen 0.21.

delivery on battery power. Delivered $V_T$ from the iVent201 decreased by 15% as a result of changing the power source from AC to battery, and $V_T$ decreased further as battery power diminished (see Fig. 6).

## Discussion

New-generation portable ventilators are smaller, more reliable, offer more advanced modes (eg, PCV, pressure support ventilation, and noninvasive positive-pressure ventilation), and create less imposed work of breathing during spontaneous breathing than did their historical counterparts.[3–6] These characteristics make portable ventilators an attractive option for providing short-term ventilation in the acute care setting and during patient transportation, and for providing long-term ventilation in subacute care facilities and in the home. In each setting, portable ventilators are expected to perform on internal power when AC power is lost or unavailable and during periods of patient movement.[7,8] The duration of portable ventilator operation depends on the supply of compressed gas and/or electricity. The maximum duration of use of any portable ventilator operating on limited supplies of gas or electricity is an important factor for clinicians to be aware of, both when making a purchasing decision and, more importantly, when using the device.

Our results show that there are significant differences in battery duration among the portable ventilators tested. In general, pneumatically driven portable ventilators have longer battery duration. Pneumatically driven portable ventilators use electricity for the flow-control valves, display, alarms, and microprocessor control board,[9–11] but no electricity is required to power the gas flow, as pressure is provided from a high-pressure piped gas source or cylinder. In addition, the battery duration of pneumatically driven portable ventilators is less affected by ventilator settings. An exception to this finding is the Uni-Vent 754, the battery duration of which is diminished by 180 min (44%) with $F_{IO_2}$ of 0.21, compared to $F_{IO_2}$ of 1.0. The Uni-Vent 754 is unique in that the drive power is changed to electric for provision of room air (electric diaphragm pump). It is likely that the duration of use of a pneumatically driven portable ventilator will be limited by the amount of compressed gas available rather than by battery duration.

Electrically driven portable ventilators use electricity for the flow-control valves, display, alarms, microprocessor control board, and to create gas pressure for flow delivery.[12–16] Within this class we tested 3 turbine ventilators (iVent201, LTV1000, and TBird AVS), 1 piston ventilator (Achieva), 1 diaphragm pump ventilator (Uni-Vent 754 on room air), and 1 piston pump ventilator (HT50). Each of these can operate without a compressed gas source, but compressed oxygen is necessary if $F_{IO_2} > 0.21$ is required. Our results suggest that turbine powered ventilators use more electricity than those that use other drive mechanisms. Different turbine powered ventilators have different operational characteristics (see Table 1). The LTV1000 uses a constant-speed turbine, and the speed is determined by the flow requirements resulting from the ventilator settings and patient ventilation requirements. The LTV1000 uses solenoid valves to control instantaneous flow output, which uses additional electricity. The TBird AVS and iVent201 use adjustable-speed turbines that vary the speed based on the instantaneous flow requirements (eg, increase during inspiration). All turbine powered ventilators provide bias flow for triggering, which further increases power consumption.

Ventilator settings have an important impact on the battery duration of electrically driven portable ventilators. PEEP and PCV have the greatest effect on the battery duration of the electrically driven ventilators. Each of the electrically driven ventilators provide internal PEEP adjustment. A dedicated solenoid is required to power the exhalation valve, which increases the battery drain as PEEP is increased. The battery duration of turbine powered ventilators is more affected by PEEP because of the higher turbine speed required to maintain the higher PEEP. The iVent201 was not capable of delivering the set $V_T$ with PEEP of 20 cm $H_2O$. However, though the delivered $V_T$ was markedly lower than the set $V_T$, the $V_T$ measured and displayed by the iVent201 did accurately reflect the $V_T$ delivered to the test lung. This ventilator would shut down with various alarms, including "sensor failure" and "mechanical failure" within a few minutes at those settings combinations. During PCV with PEEP of 20 cm $H_2O$, the iVent201 would enter into an "open loop" ventilation mode that would allow spontaneous breathing. During VCV with 20 cm $H_2O$ PEEP, the iVent201 exhibited a decreased $V_T$ delivery when the power source was changed from AC to battery power, and $V_T$ further decreased as battery power diminished (see Fig. 6). The mean battery duration of the iVent201 is probably overestimated, as battery duration at the settings that include 20 cm $H_2O$ PEEP are not included and would certainly shorten the mean duration. The iVent201 should not be used clinically with patients suffering low lung compliance requiring high PEEP. The use of PCV causes an additional drain on the batteries of electrically driven ventilators because of the higher flow delivery requirement during early inspiration. The HT50 behaved differently than the other electrically driven ventilators during PCV: the battery duration was higher with PCV than with VCV. This may be due to an inherent flow limitation of the HT50 and the fact that there is no flow at the end of inspiration. The drive mechanism of the HT50 stops operating when no flow is required, whereas the other ventilators still deliver pressure to the circuit at end-inspiration, even though flow to the patient is zero.

The battery duration of the ventilators we studied is minimally affected by the $F_{IO_2}$ setting. With these ventilators (except HT50), $F_{IO_2}$ is titrated with a series of high-pressure proportioning valves that require electricity. Testing the effect of $F_{IO_2}$ on battery duration was done only at the extreme settings (0.21 and 1.0), and the effect of the $F_{IO_2}$ setting is hypothesized to be linear (ie, battery duration shortens as $F_{IO_2}$ is increased) because of the more frequent valve operation as $F_{IO_2}$ is increased. Low-pressure oxygen can also be bled into the ventilator or breathing circuit to increase $F_{IO_2}$, which would not affect the battery duration but would cause less accurate control of $F_{IO_2}$.[4]

The size and weight of portable ventilators are important characteristics. Clinicians desire small, lightweight units with long battery duration, but, in general, larger and heavier units have the longest battery duration. An exception to that generalization is the TBird AVS, which is the largest and heaviest ventilator tested and yet has the shortest battery duration. Manufacturers must compromise in designing ventilators, based on the intended application. For example, a ventilator designed for use in the acute care setting will probably require less battery duration, because in that setting there are numerous electric outlets and a backup generator within the institution. Battery use in that setting is necessary only during brown-outs and patient transport. The average duration of patient transport in an acute care hospital is roughly 50–75 min.[17,18] However, battery power is not required for the entire transport, since the ventilator can use AC power at the transport destination. The average duration of patient transport (one way) to the destination is 5–40 min, and the average time spent at the destination is 35 min.[18,19] The average duration of interhospital patient transport is 4 hours and 23 min.[20] Although it has not been specifically reported, the average time spent moving the patient from bed to transport vehicle (eg, ambulance or helicopter) is probably similar to that experienced during intrahospital transport (10–40 min). The main difference is that a limited supply of compressed gas and electricity are available when ventilating the patient in the transport vehicle.[21,22] Portable ventilators for ventilator-dependent patients outside the acute care setting require longer battery duration. Extended battery duration in that setting may improve the patient's quality of life. Many of the portable ventilators we tested have optional direct current power converters and external batteries that would extend their duration of use on battery power. Our testing was limited to internal batteries only.

Most portable ventilators use sealed lead acid batteries because they are durable, inexpensive, do not exhibit voltage memory, and function safely independent of position. Their duration of use depends on their capacity (size) and the load placed on them. Energy provided by sealed lead acid batteries is not finite. For example, a 100 amp-hour battery rated at 20 hours implies that 100 amp-hours are available for 20 hours. Using a load of 5 amp/h will result in a battery duration of 20 hours ($20 \times 5 = 100$ amp-hours). At higher loads, however, the number of amp-hours available from the battery decreases. If the battery mentioned above is drawn down over 1 hour there would only be 59 amp-hours available (decreased capacity with increased load).

Limitations of the present study include the fact that, in general, only one of each device was tested. Although we observed minimal variation ($< 5\%$ total battery duration) between like models of portable ventilators used in this study (Avian, Uni-Vent 750, iVent201, and LTV1000), it was not a primary end point and our test methods were not designed to evaluate this variability. Testing was performed in the laboratory setting; the battery duration of each device will probably be shorter in clinical use. One reason for this is that to conserve battery power portable ventilators shut down the monitor display after a few minutes of no activity. We allowed each ventilator's monitor to remain idle during the test period. Also, room temperature was constant (24° C) during all testing, and each ventilator was placed on a stand or tabletop during testing. In clinical use the operating temperature is likely to be higher if the device is placed in the bed with the patient and air flow around the ventilator (and, thus, heat dissipation) is limited by close proximity to pillows, sheets, and other equipment.

## Conclusions

Battery duration is an important characteristic of portable ventilators. Clinicians should be aware of the battery duration characteristics of any ventilator they use and should recognize that the battery duration may be substantially shorter than that listed in the operator's manual. The largest deviation from the manufacturer-specified battery duration was 67% shorter battery duration, due to the ventilator settings and lung impedance conditions. A self-inflating, manual resuscitator should be a mandatory and standard piece of equipment accompanying any mechanically ventilated patient during transport, in case of ventilator malfunction.[7] Knowledge of battery duration characteristics and the effects of various ventilator settings may facilitate avoidance of equipment failure and adverse outcomes.

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Special Articles

# Chronic Obstructive Pulmonary Disease Surveillance—United States, 1971–2000

David M Mannino MD, David M Homa PhD, Lara J Akinbami MD,
Earl S Ford MD, and Stephen C Redd MD

**Introduction**
**Methods**
  **Self-Reported Prevalence**
  **Objectively Determined Prevalence**
  **Activity and Functional Limitations**
  **Physician Office Visits, Hospital Outpatient Department Visits, and Emergency Department Visits**
  **Hospitalizations**
  **Mortality**
**Results**
  **Prevalence**
  **Activity and Functional Limitations**
  **Physician Office and Hospital Outpatient Department Visits**
  **Emergency Department Visits**
  **Hospitalizations**
  **Deaths**
**Discussion**

**PROBLEM/CONDITION: Chronic obstructive pulmonary disease (COPD) includes chronic bronchitis and emphysema but has been defined recently as the physiologic finding of nonreversible pulmonary function impairment. This surveillance summary reports trends in different measures of COPD during 1971–2000. REPORTING PERIOD COVERED: This report presents national data regarding objectively determined COPD (1971–1994); COPD-associated activity and functional limitations (1980–1996); self-reported COPD prevalence, COPD physician office and hospital outpatient department visits, COPD hospitalizations, and COPD deaths (1980–2000); and COPD emergency department visits (1992–2000). DESCRIPTION OF SYSTEMS: The Centers for Disease Control's National Center for Health Statistics conducts the National Health Interview Survey annually, which includes questions concerning COPD and activity limitations. The National Center for Health Statistics collects physician office-visit data in the National Ambulatory Medical Care Survey, emergency department and hospital outpatient department data in the National Hospital Ambulatory Medical Care Survey, hospitalization data in the National Hospital Discharge Survey, and death data in the Mortality Component of the National Vital Statistics System. Data regarding pulmonary function were obtained from the National Health and Nutrition Examination Surveys (NHANES) I (1971–1975) and III (1988–1994), and data regarding functional limitation were obtained from NHANES III, Phase 2 (1991–1994). RESULTS: During 2000, an estimated 10 million U.S. adults reported physician-diagnosed COPD. However, data from NHANES III estimate that approximately 24 million United States adults have evidence of impaired lung function, indicating that COPD is underdiagnosed. During 2000, COPD was responsible for 8 million physician office and hospital outpatient visits, 1.5 million emergency department visits, 726,000 hospitalizations, and**

**119,000 deaths. During the period analyzed, the most substantial change was the increase in the COPD death rate for women, from 20.1/100,000 in 1980 to 56.7/100,000 in 2000, compared with the more modest increase in the death rate for men, from 73.0/100,000 in 1980 to 82.6/100,000 in 2000. In 2000, for the first time, the number of women dying from COPD surpassed the number of men dying from COPD (59,936 vs 59,118). Another substantial change observed is that the proportion of the population aged < 55 years with mild or moderate COPD, on the basis of pulmonary function testing, decreased from 1971–1975 to 1988–1994, possibly indicating that the upward trends in COPD hospitalizations and mortality might not continue. INTERPRETATION: COPD is a major cause of morbidity, mortality, and disability in the U.S. Despite its ease of diagnosis, COPD remains an underdiagnosed disease, chiefly in its milder and more treatable form.** *Key words: chronic obstructive pulmonary disease, COPD, bronchitis, emphysema, pulmonary, survey.* [Respir Care 2002;47(10):1184–1199]

## Introduction

Chronic obstructive pulmonary disease (COPD) is a group of diseases characterized by air flow obstruction that can be associated with breathing-related symptoms (eg, chronic cough, exertional dyspnea, expectoration, and wheeze).[1] COPD can be present with or without substantial physical impairment or symptoms, and it is the fourth leading cause of death in the United States.[2] However, COPD is often a silent and unrecognized disease, chiefly in its early phases.[3] During 1993, the estimated direct medical costs of COPD were $14.7 billion.[4] Also during 1993, the estimated indirect cost related to morbidity (eg, loss of work time and productivity) and premature mortality was an additional $9.2 billion, for a total of $23.9 billion. *Healthy People 2010* includes 2 objectives related to COPD: to reduce the proportion of adults whose activity is limited because of chronic lung and breathing problems to 1.5% (Objective 24–9) and to reduce deaths from COPD among adults aged ≥ 45 years to 60 deaths/100,000 (Object 24–10).[5]

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Traditionally, COPD has been diagnosed on the basis of patient-reported symptoms.[6,7] The recently published definition from the Global Initiative for Chronic Obstructive Lung Disease (GOLD) has classified COPD as "a disease state characterized by air flow limitation that is not fully reversible"[8] and recommends measurement of lung function both to diagnose disease and categorize disease severity. Air flow limitation is the slowing of expiratory air flow as measured by spirometry, with a persistently low forced expiratory volume in the first second ($FEV_1$) and a low $FEV_1$/forced vital capacity (FVC) ratio despite treatment.[7] The GOLD criteria for mild COPD (stage 1) is an $FEV_1$/FVC ratio of $< 70\%$ and $FEV_1$ of $> 80\%$ predicted, and the criteria for moderate COPD (stage 2 or 3) is an $FEV_1$/FVC ratio of $< 70\%$ and an $FEV_1 \leq 80\%$ predicted.[8,9]

This report presents national data regarding objectively determined (ie, by spirometry) COPD (1971–1994); COPD-associated activity and functional limitations (1980–1996); self-reported COPD prevalence, COPD physician office and hospital outpatient department visits, COPD hospitalizations, and COPD deaths (1980–2000); and COPD emergency department visits (1992–2000).

## Methods

For this report, we defined COPD as including chronic bronchitis and emphysema when we used survey data based on clinical diagnosis. In doing so, asthma and bronchiectasis were excluded, but they are often included in other definitions of COPD and related conditions. Asthma was excluded because its etiology and treatment differs from

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that for COPD, although clinical similarities exist between these conditions. Centers for Disease Control (CDC) recently published surveillance data for asthma.[10] Bronchiectasis was excluded because it typically is caused by infection and has a different treatment and prognosis than COPD.

Data from national health surveys conducted by CDC's National Center for Health Statistics (NCHS) were used to report these measures. In addition, the resident population estimates from the U.S. Bureau of the Census were used as denominators for rates of COPD office visits, COPD emergency department visits, COPD hospitalizations, and COPD deaths. These denominators vary slightly from those typically used with these databases because we wanted a consistent denominator for these measures. The National Health and Nutrition Examination Surveys (NHANES) and National Health Interview Survey (NHIS) weights (corresponding to the civilian, noninstitutionalized population of the U.S.) were used to estimate the population when those data sets were analyzed. When calculating rates that used pulmonary function data, we limited the denominator to subjects who had pulmonary function testing (PFT) performed. In the tables for this report, annual estimates are listed for selected years (1980, 1985, 1990, and 1995–2000); in the figures, annual estimates are listed for 1980–2000 for the majority of measures. Measures that were only available for limited years included: (1) activity limitation, for which years are grouped because the denominator (persons with COPD) was smaller; (2) objectively determined COPD, for which 2 samples (1971–1975 and 1988–1994) were available; and (3) functional limitation, for which 1 sample (1991–1994) was available.

Our results were age-adjusted to the 2000 U.S. population by using 5 age groups (ie, 25–44 years, 45–54 years, 55–64 years, 65–74 years, and ≥ 75 years). Data were stratified by sex, race (ie, white, black, and other), and age group (ie, 25–44 years, 45–54 years, 55–64 years, 65–74 years, and ≥ 75 years) for subgroup analyses. Persons of other races (eg, Asians and American Indians) were grouped together because their numbers in individual racial groups were too limited to yield statistically stable estimates. We analyzed all data by using SAS[11] and SUDAAN software.[12] Two-tailed, weighted, least squares regression (by using the inverse of the relative standard error of the estimate as the weight) was used to determine whether linear trends were statistically significant. Trend testing was performed only when ≥ 5 years of continuous data were available for analysis. Two-tailed *t* tests were used to compare measures between racial groups, age groups, and males and females. By using the Bonferroni adjustment for multiple comparison among ≤ 5 groups, we considered a familywide p value of ≤ 0.05 as statistically significant.

### Self-Reported Prevalence

The National Center for Health Statistics conducts NHIS annually among a probability sample of the civilian, noninstitutionalized population of the U.S.[13] Before 1997, for one sixth of the NHIS-sampled households (approximately 20,000 of 120,000 persons), respondents were asked whether anyone in the household had any of 17 chronic respiratory conditions, including chronic bronchitis or emphysema, during the preceding 12 months. Under this design, information related to COPD among adults might have been reported by the respondents themselves or by a household respondent. A COPD diagnosis was determined if the response was positive to either of the following questions: "During the past 12 months did [you] have bronchitis," if the condition was listed as chronic, or "During the past 12 months did [you] have emphysema?"

Beginning in 1997, NHIS collected information concerning COPD for a randomly selected adult in each household. COPD prevalence was determined if participants responded positively to either of the following questions: "Have you ever been told by a doctor or other health professional that you had emphysema," or "During the past 12 months, have you been told by a doctor or other health professional that you had chronic bronchitis?".[13]

NHANES III was conducted during 1988–1994.[12] Subjects were asked, "Has a doctor ever told you that you had chronic bronchitis"; "Has a doctor ever told you that you had emphysema"; and "Do you still have chronic bronchitis?" Subjects were regarded as having COPD if they reported a diagnosis of emphysema or current chronic bronchitis. SUDAAN was used to determine relative standard errors (RSEs) of the estimates and to indicate which estimates were reliable (ie, RSE < 30%).[14]

### Objectively Determined Prevalence

During 1971–1975, NCHS conducted the first National Health and Nutrition Examination Survey (NHANES I).[15,16] Both NHANES I and NHANES III were probability samples of the civilian, noninstitutionalized U.S. population. Spirometry was obtained on a subset of survey participants (5,080 adults in NHANES I and 13,869 adults in NHANES III). The procedures used have been documented previously.[12,16] Values used in this analysis included FVC, $FEV_1$, and the $FEV_1$/FVC ratio. Predicted values of $FEV_1$ and FVC were identified by performing linear regression (stratified by sex and by using age and height as predictors) on a subgroup of participants who were white and who had never smoked and did not report respiratory symptoms or physician-diagnosed lung disease. Results from these regression models were applied to the data from all participants to obtain predicted values of $FEV_1$ and FVC. An adjustment factor of 0.88 was used to estimate predicted values for black participants.[17] Participants were classified as having moderate obstructive lung disease if both the $FEV_1/FVC$ ratio was $< 70\%$ and the $FEV_1$ was $\leq 80\%$ of the predicted value.[8] Participants were classified as having mild obstructive lung disease if the $FEV_1/FVC$ ratio was $< 70\%$ and the $FEV_1$ was $> 80\%$ of the predicted value.

### Activity and Functional Limitations

Through 1996, NHIS provided data related to activity limitations overall and resulting from specific conditions. Activity limitations were classified into 1 of 4 groups: unable to perform major activity; certain limitation in major activity; limitation in other activities; or no activity was limited. Major activity was defined as working or keeping house for adults aged 18–69 years, and ability to execute activities of independent living for those aged ≥ 70 years. The percentage of persons with and without COPD who reported activity limitation (including unable to perform major activities, limited in major activities, and limited in other activities) was calculated for all adults aged ≥ 25 years. The percentage of persons who reported having COPD and who also reported COPD-associated activity limitation was also calculated. Respondents were categorized as limited because of COPD if they reported that COPD was the primary or secondary cause of the limitation. Because of the relatively low number of persons reporting limited activity, multiple years of data were used to obtain stable estimates.

Data from NHANES III, Phase 2 (1991–1994), were used to determine physical functional limitation. During the home interview, participants aged ≥ 17 years were asked a series of questions regarding their physical functioning. From these questions, the following 3 items were selected: difficulty walking ¼ mile; lifting or carrying something weighing 10 pounds; and need of help from other persons in handling routine tasks (eg, doing everyday household chores, conducting necessary business, shopping, or getting around for other purposes). For the first 2 items (difficulty walking or lifting), respondents could answer "no difficulty," "some difficulty," "much difficulty," and "unable to do." These responses were dichotomized into no difficulty and any difficulty. For the last item (handling routine needs), respondents could respond yes or no. The age-adjusted proportion of functional limitation among participants with current reported COPD (chronic bronchitis or emphysema) and no reported COPD were identified,[3] and this was repeated for the same group of participants stratified by level of pulmonary function impairment. SUDAAN was used to determine RSEs of the estimates.[14]

### Physician Office Visits, Hospital Outpatient Department Visits, and Emergency Department Visits

Ambulatory medical care is the predominant means of providing health care services in the U.S. We considered both physician office visits and hospital outpatient department visits, which are collected, by using different surveys, as office visits and emergency department visits separately. Physician office-visit data were collected through the National Ambulatory Medical Care Survey (NAMCS), which NCHS conducted during 1973–1981, 1985, and annually since 1989.[18] Approximately 2,000 physicians participated each year, reporting data concerning approximately 30,000 patient encounters. Hospital outpatient-visit data and emergency department-visit data were collected by using the National Hospital Ambulatory Medical Care Survey (NHAMCS), which has been conducted annually since 1992.[19] Approximately 500 hospitals are sampled each year, resulting in approximately 30,000 outpatient department encounters and 30,000 emergency department encounters.

By using both data sets, we identified all patient visits for which COPD (International Classification of Diseases, 9th revision, Clinical Modification (ICD-9-CM), codes 490–492, 496)[20] was the first-listed diagnosis. Sample weights were used to obtain national estimates of annual outpatient visits (physician office and hospital outpatient department visits were combined beginning in 1992) and emergency department visits for COPD. RSEs, which are listed with the database documentation, were used to determine which estimates were reliable (ie, RSE < 30%).[21]

### Hospitalizations

The National Hospital Discharge Survey (NHDS), conducted annually by NCHS since 1965, is a national survey of approximately 275,000 patient records from approximately 500 nonfederal general and short-stay specialty hospitals. A hospitalization for COPD was defined as a primary discharge diagnosis of COPD (ICD-9-CM codes 490–492, 496).[20] In addition, a hospitalization was considered as a result of COPD if it had a first-listed diagnosis of acute bronchitis (ICD-9-CM code 466–466.1) accompanied by another listed diagnosis of COPD. During 1980–1982, approximately 15%–24% of all first-listed diagnoses of acute bronchitis were accompanied by another listed diagnosis of COPD (chronic bronchitis, emphysema, or COPD not specified); however, during 1983–1991, this proportion ranged from 39% to 58%. During 1992, a new ICD-9-CM code, 491.21, was introduced to categorize obstructive chronic bronchitis with acute exacerbation. After the introduction of this new code, 3%–10% of all first-listed diagnoses of acute bronchitis were accompanied by

Table 1. Estimated Number of Persons With Self-Reported, Lifetime Emphysema or Chronic Bronchitis During the Preceding 12 Months (1980–1996) or Self-Reported, Physician-Diagnosed Lifetime Emphysema or Chronic Bronchitis During the Preceding 12 Months (1997–2000), by Race, Sex, and Age Group—United States, National Health Interview Survey, 1980–2000*

| Variable | 1980 | 1985 | 1990 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|---|---|---|---|---|---|---|---|---|---|
| Race | | | | | | | | | |
| White | 6,623,000 | 7,944,000 | 9,087,000 | 9,524,000 | 8,837,000 | 9,408,000 | 8,968,000 | 8,587,000 | 9,178,000 |
| Black | 395,000 | 680,000 | 664,000 | 905,000 | 959,000 | 1,175,000 | 891,000 | 778,000 | 976,000 |
| Other | 125,000† | 68,000† | 87,000† | 264,000 | 314,000 | 344,000 | 294,000 | 337,000 | 361,000 |
| Sex | | | | | | | | | |
| Male | 3,477,000 | 3,186,000 | 3,640,000 | 3,867,000 | 3,788,000 | 4,083,000 | 3,718,000 | 3,576,000 | 3,798,000 |
| Female | 3,665,000 | 5,505,000 | 6,197,000 | 6,825,000 | 6,323,000 | 6,844,000 | 6,434,000 | 6,126,000 | 6,717,000 |
| Age group (y) | | | | | | | | | |
| 25–44 | 2,090,000 | 3,122,000 | 3,583,000 | 4,121,000 | 3,908,000 | 3,670,000 | 3,173,000 | 3,087,000 | 3,157,000 |
| 45–54 | 1,149,000 | 1,205,000 | 1,498,000 | 2,071,000 | 1,877,000 | 2,043,000 | 2,055,000 | 1,811,000 | 2,184,000 |
| 55–64 | 1,500,000 | 1,631,000 | 1,590,000 | 1,674,000 | 1,711,000 | 1,845,000 | 1,748,000 | 1,725,000 | 1,879,000 |
| 65–74 | 1,749,000 | 1,811,000 | 2,023,000 | 1,683,000 | 1,470,000 | 2,021,000 | 1,871,000 | 1,639,000 | 1,721,000 |
| ≥ 75 | 655,000 | 921,000 | 1,143,000 | 1,144,000 | 1,145,000 | 1,348,000 | 1,306,000 | 1,439,000 | 1,573,000 |
| Total§ | 7,143,000 | 8,690,000 | 9,837,000 | 10,693,000 | 10,111,000 | 10,927,000 | 10,153,000 | 9,701,000 | 10,515,000 |

*All relative standard errors are < 30%, unless otherwise indicated

†Relative standard error of the estimate is 30%–50%, the estimate is unreliable.

§Numbers for each variable might not add to total because of rounding

Table 2. Estimated Annual Prevalence* of Self-Reported, Lifetime Emphysema or Chronic Bronchitis During the Preceding 12 Months, (1980–1996) or Self-Reported, Physician-Diagnosed Lifetime Emphysema or Chronic Bronchitis During the Preceding 12 Months (1997–2000), by Race, Sex, and Age Group—United States, National Health Interview Survey, 1980–2000†

| Variable | 1980 | 1985 | 1990 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|---|---|---|---|---|---|---|---|---|---|
| Race§ | | | | | | | | | |
| White | 58.9¶ | 63.4 | 67.5 | 67.2 | 62.1 | 65.7 | 62.4 | 59.4 | 63.6 |
| Black | 31.2¶ | 45.8 | 39.7 | 50.3 | 52.0 | 63.2 | 47.4 | 40.8 | 50.4 |
| Other | 46.3** | 19.5** | 17.4** | 39.8 | 42.5 | 40.0 | 31.0 | 33.0 | 31.4 |
| Sex§ | | | | | | | | | |
| Male | 58.0 | 47.3¶ | 49.2¶ | 48.9¶ | 47.3¶ | 50.3¶ | 45.4¶ | 43.3¶ | 45.5¶ |
| Female | 53.9 | 72.3¶ | 75.2¶ | 78.2¶ | 71.6¶ | 76.7¶ | 71.5¶ | 67.3¶ | 73.2¶ |
| Age group (y) | | | | | | | | | |
| 25–44 | 34.6 | 43.4 | 44.8 | 49.6 | 46.9 | 44.0 | 38.2 | 37.4 | 38.5 |
| 45–54 | 51.2 | 53.8 | 59.2 | 67.0 | 58.4 | 61.1 | 59.8 | 50.9 | 59.2 |
| 55–64 | 71.2 | 73.7 | 74.8 | 80.5 | 81.4 | 85.9 | 78.4 | 74.9 | 79.5 |
| 65–74 | 113.6 | 108.5 | 111.8 | 90.7 | 79.8 | 111.2 | 103.8 | 92.0 | 96.4 |
| ≥ 75 | 75.3 | 89.0 | 97.8 | 88.4 | 85.8 | 97.4 | 92.0 | 98.1 | 106.0 |
| Total§ | 55.8 | 60.5 | 62.9 | 64.3 | 60.1 | 64.2 | 59.1 | 55.9 | 60.0 |

*Per 1,000 population

†All relative standard errors are < 30%, unless otherwise indicated

§Age-adjusted to 2000 U.S. population

¶Represents a statistically significant difference between blacks and whites or males and females for that year

**Relative standard error of the estimate is 30%–50%; the estimate is unreliable

another listed diagnosis of COPD during 1992–2000. During any selected year, race was missing for 5%–20% of the sample.[22] These persons were excluded from the race-specific rate calculations but were included in all other rate calculations. Published relative standard errors[21] were used to indicate which estimates were reliable (ie, RSE < 30%).

### Mortality

The Mortality Component of the National Vital Statistics System includes medical conditions and reported demographic characteristics regarding death.[23] We searched for deaths for which COPD was the underlying cause (ICD-9 codes 490–492, 496, for 1980–1998; ICD-10 codes

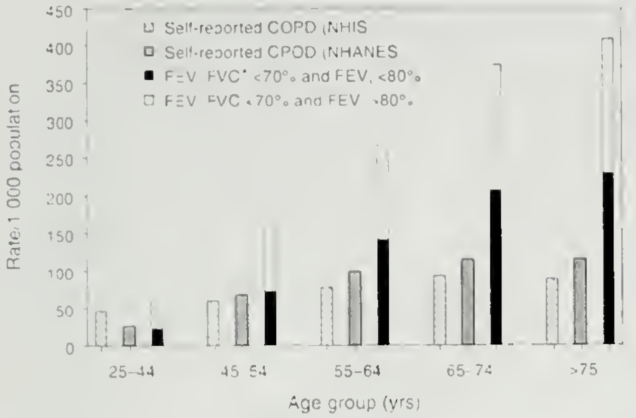

Fig. 1. Estimated prevalence of self-reported chronic obstructive pulmonary disease (COPD), by age group, United States, from questionnaire data from the National Health Interview Survey (NHIS), 1988–1994, and questionnaire and pulmonary function data from the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994. $FEV_1$ = forced expiratory volume in the first second. FVC = forced vital capacity.

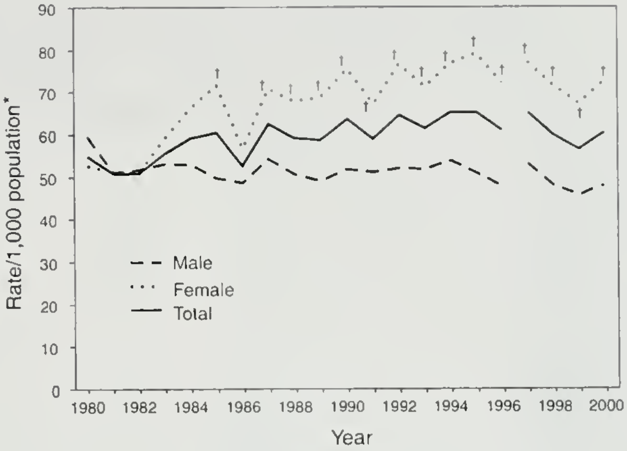

Fig. 2. Estimated annual prevalence of self-reported chronic obstructive pulmonary disease (COPD), by sex and year, United States, from the National Health Interview Survey, 1980–2000. * Age-adjusted to 2000 U.S. population. † Represents a statistically significant difference from rate among males.

J40-J44 for 1999–2000).[24,25] In ICD-10, the term chronic lower respiratory disease is used to describe the diseases encompassed by the term COPD. Comparability ratios for ICD-10 relative to ICD-9 for COPD are as follows: 0.97 for emphysema; 1.10 for other chronic lower respiratory diseases; 0.39 for chronic and unspecified bronchitis; and 1.05 for all chronic lower respiratory diseases, which includes asthma.[26] Standard errors were calculated as the square root of the inverse of the number of deaths.[23]

## Results

### Prevalence

The estimated number and rates of persons reporting COPD are included in this report (Tables 1 and 2) (Figures 1-3). Whites had statistically significant higher rates than blacks for certain years examined (Fig. 2). Since 1987, women have had higher rates of self-reported COPD than men, and during 1980–1996 the trend for COPD increased for women but not for men (Fig. 3). The 1997 redesign of NHIS might have had an effect on COPD prevalence, although the observed year-to-date variation in COPD prevalence makes this difficult to discern (Figs. 2 and 3).

Data from NHANES I and NHANES III estimating the prevalence of COPD on the basis of spirometric definitions are presented (Tables 3 and 4) (Fig. 1). For both mild and moderate COPD, the prevalence was higher among men than women and increased with increasing age. From NHANES I to NHANES III, prevalence of moderate COPD decreased among men but not among women (Table 4). When stratified by age group, a statistically significant decrease occurred in moderate COPD among persons aged 25–54 years, but not among other groups; and, when stratified by race, a similar decrease occurred among blacks, but not whites.

### Activity and Functional Limitations

The age-adjusted employment rate is lower among persons who report they have COPD (Table 5). During the period analyzed, only limited change occurred in prevalence of activity limitation among adults with self-reported COPD (Table 5). Adults with COPD have a prevalence of any activity limitation approximately twice as high as adults

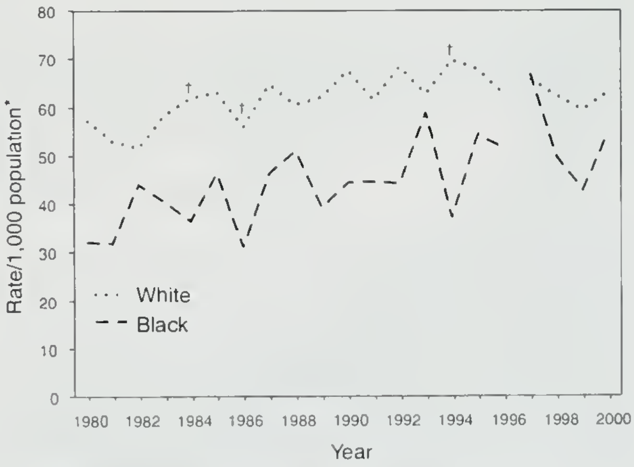

Fig. 3. Estimated annual prevalence of self-reported chronic obstructive pulmonary disease (COPD), by race and year, United States, from the National Health Interview Survey, 1980–2000. * Age-adjusted to 2000 U.S. population. † Represents a statistically significant difference from rate among blacks.

Table 3. Estimated Number of Persons with Evidence of Either Mild or Moderate Obstructive Lung Disease, by Race, Sex, and Age Group—United States, National Health and Nutrition Examination Surveys I, 1971–1975, and III, 1988–1994*

| Variable | Mild Obstructive Lung Disease ($FEV_1/FVC$† < 70% and $FEV_1 \geq 80\%$ Predicted) | | Moderate Obstructive Lung Disease ($FEV_1/FVC$ < 70% and $FEV_1 < 80\%$ Predicted) | |
|---|---|---|---|---|
| | 1971–1975 | 1988–1994 | 1971–1975 | 1988–1994 |
| Race | | | | |
| White | 5,626,000 | 10,750,000 | 6,130,000 | 10,882,000 |
| Black | 814,000 | 843,000 | 635,000 | 893,000 |
| Other | —§ | 455,000¶ | —§ | 317,000¶ |
| Sex | | | | |
| Male | 3,668,000 | 7,181,000 | 4,453,000 | 6,401,000 |
| Female | 2,823,000 | 4,867,000 | 2,357,000 | 5,691,000 |
| Age group (y) | | | | |
| 25–44 | 1,989,000 | 2,890,000 | 1,800,000 | 1,799,000 |
| 45–54 | 1,737,000 | 2,095,000 | 1,672,000 | 1,741,000 |
| 55–64 | 1,637,000 | 2,503,000 | 1,870,000 | 2,786,000 |
| 65–74 | 1,128,000 | 2,760,000 | 1,468,000 | 3,447,000 |
| ≥ 75 | — | 1,801,000 | — | 2,318,000 |
| Total¶** | 6,491,000 | 12,049,000 | 6,810,000 | 12,092,000 |

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*All relative standard errors are < 30%, unless otherwise indicated.

†$FEV_1$ is the forced expiratory volume in the first second, and FVC is the forced vital capacity.

§Relative standard error of the estimate exceeds 50%.

¶Relative standard error of the estimate is 30%–50%; the estimate is unreliable.

**Numbers for each variable might not add to total because of rounding error.

Table 4. Estimated Prevalence* of Either Mild or Moderate Obstructive Lung Disease, by Race, Sex, and Age Group—United States, National Health and Nutrition Examination Surveys I, 1971–1975, and III, 1988–1994†

| Variable | Mild Obstructive Lung Disease ($FEV_1/FVC$§ < 70% and $FEV_1 \geq 80\%$ Predicted) | | Moderate Obstructive Lung Disease ($FEV_1/FVC$ < 70% and $FEV_1 < 80\%$ Predicted) | |
|---|---|---|---|---|
| | 1971–1975 | 1988–1994 | 1971–1975 | 1988–1994 |
| Race*¶ | | | | |
| White | 70.6 | 70.8 | 76.5 | 67.1 |
| Black | 112.3** | 50.2** | 87.5** | 55.8** |
| Other | —†† | 80.0§§ | —†† | 52.8 |
| Sex*¶ | | | | |
| Male | 88.8 | 90.9 | 108.1** | 74.3** |
| Female | 60.9 | 48.7 | 50.8 | 58.2 |
| Age group (y) | | | | |
| 25–44 | 48.9** | 36.8** | 44.3** | 22.9** |
| 45–54 | 101.1 | 87.1 | 97.3** | 72.4** |
| 55–64 | 123.2 | 126.2 | 140.7 | 140.5 |
| 65–74 | 133.5 | 165.4 | 173.8 | 206.6 |
| ≥ 75 | — | 178.2 | — | 229.3 |
| Total*¶ | 73.9 | 69.0 | 77.4 | 65.7 |

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*Per 1,000 population; limited to ages 25–74 years.

†All relative standard errors are < 30%, unless otherwise indicated.

§$FEV_1$ is the forced expiratory volume in the first second, and FVC is the forced vital capacity.

¶Age-adjusted to 2000 U.S. population.

**Represents a statistically significant difference between the National Health and Nutrition Examination Surveys I and III for that subgroup.

††Relative standard error of the estimate exceeds 50%.

§§Relative standard error of the estimate is 30%–50%; the estimate is unreliable.

Table 5. Average Annual Percentage of Persons Who Report Being Employed, by Self-Reported Chronic Obstructive Pulmonary Disease (COPD) Status and Percentage With Activity Limitation, by Self-Reported COPD Status: Adults Aged ≥ 25 years—United States, National Health Interview Survey, 1980–1996*†

| Variable | 1980–1982 | 1985–1987 | 1990–1992 | 1994–1996 |
|---|---|---|---|---|
| Adults who report being employed (%) | | | | |
| Adults with self-reported COPD | 49.7 | 53.8 | 57.8 | 57.5 |
| Adults without self-reported COPD | 59.7 | 62.2 | 64.0 | 64.6 |
| Adults with any activity limitation (%) | | | | |
| Adults with self-reported COPD | 44.1 | 37.4 | 36.8 | 38.6 |
| Adults without self-reported COPD | 20.0 | 18.5 | 18.4 | 18.4 |
| Adults with COPD who reported COPD-associated activity limitation (%) | | | | |
| All | 14.1 | 10.4 | 8.4 | 8.0 |

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*Relative standard errors are < 30%, unless otherwise indicated

†Rates adjusted to the 2000 population of adults aged ≥ 25 years.

Table 6. Percentage of Subjects Reporting Functional Limitations, by Chronic Obstructive Pulmonary Disease (COPD) Status, Defined by Self-Report or on the Basis of Pulmonary Function Tests Among Adults Aged ≥ 25 Years—United States, National Health and Nutrition Examination Survey III, Phase 2, 1991–1994*

| Variable | *N* | Weighted Percentage | Difficulty Walking ¼ Mile (%)† | Difficulty Lifting or Carrying 10 Pounds (%)† | Need Help in Handling Routine Needs (%)† |
|---|---|---|---|---|---|
| Self-reported COPD | | | | | |
| No | 6,289 | 94.8 | 11.2§ | 9.5§ | 2.4 |
| Yes | 311 | 5.2 | 34.2§ | 30.5§ | 5.9 |
| COPD, based on pulmonary function tests | | | | | |
| $\text{FEV}_1$/FVC¶ > 70% (ie, normal) | 5,581 | 84.8 | 12.0 | 10.0 | 2.8 |
| $\text{FEV}_1$/FVC ≤ 70% and $\text{FEV}_1$ ≥ 80% (ie, mild) | 488 | 7.3 | 10.9 | 11.2 | 1.2 |
| $\text{FEV}_1$/FVC ≤ 70% and $\text{FEV}_1$ < 80% (ie, moderate) | 531 | 7.8 | 18.0 | 13.9 | 7.0** |

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*All relative standard errors are < 30%

†Age-adjusted to population distribution of the National Health and Nutrition Examination Survey III sample.

§Represents a statistically significant difference between subjects with and without COPD

¶$\text{FEV}_1$ is the forced expiratory volume in the first second, and FVC is the forced vital capacity

**Relative standard error of the estimate is 30%–50%; the estimate is unreliable.

without COPD. During 1994–1996, 8.0% of persons with COPD reported activity limitation caused by their COPD.

Respondents with self-reported COPD had a higher proportion of functional limitation than did persons without self-reported COPD (Table 6). Among subjects with spirometrically determined COPD, no measures of functional impairment were substantially increased, compared with subjects with normal lung function (Table 6).

## Physician Office and Hospital Outpatient Department Visits

Data for physician office and hospital outpatient department visits for COPD are presented in this report (Tables 7 and 8). No statistically significant trend during 1989–2000 was identified (Fig. 4). Whites had a significantly higher rate of visits than did blacks during certain years examined, although estimates for blacks were unreliable for the majority of years. No consistent differences were identified between men and women for physician office and hospital outpatient department visits for COPD, but visit rates increased with increasing age.

## Emergency Department Visits

Data for emergency department visits for COPD are presented (Tables 9 and 10). During the study period, a significant upward trend occurred in emergency department visits for COPD. Blacks had consistently higher rates than whites for the majority of years examined (Fig. 5). No consistent pattern for sex was observed. Within age groups, the highest rates were observed among older age groups.

## Hospitalizations

Data for hospitalizations for COPD are presented (Tables 11 and 12). During 1984–1989, hospitalization rates

Table 7. Estimated Annual Number of Physician Office Visits (1980–1998) and Hospital Outpatient Visits (1995–1998) for Chronic Obstructive Pulmonary Disease as the First-Listed Diagnosis, by Race, Sex, and Age Group—United States, National Ambulatory Medical Care Survey, 1980–2000, and National Hospital Ambulatory Medical Care Survey, 1995–2000[*]

| Variable | 1980 | 1985 | 1990 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|---|---|---|---|---|---|---|---|---|---|
| Race | | | | | | | | | |
| White | 4,964,000 | 7,336,000 | 9,099,000 | 10,622,000 | 8,650,000 | 9,245,000 | 13,223,000 | 9,138,000 | 6,996,000 |
| Black | 484,000[†] | 387,000[†] | 525,000[†] | 472,000[†] | 931,000[†] | 631,000[†] | 678,000[†] | 1,106,000 | 614,900[†] |
| Other | —[§] | —[§] | 957,000[†] | 398,000[†] | 348,000[†] | —[§] | —[§] | —[§] | —[§] |
| Sex | | | | | | | | | |
| Male | 2,852,000 | 3,934,000 | 4,883,000 | 5,906,000 | 4,881,000 | 5,908,000 | 6,501,000 | 4,275,000 | 3,956,000 |
| Female | 2,664,000 | 3,942,000 | 5,698,000 | 5,587,000 | 5,047,000 | 4,900,000 | 7,697,000 | 6,080,000 | 4,041,000 |
| Age group (y) | | | | | | | | | |
| 25–44 | 1,462,000 | 1,936,000 | 3,109,000 | 2,651,000 | 2,215,000 | 2,605,000 | 3,221,000 | 1,784,000 | 1,446,000 |
| 45–54 | 659,000 | 869,000 | 1,270,000 | 1,334,000 | 1,147,000 | 1,588,000 | 1,616,000 | 1,294,000 | 1,182,000 |
| 55–64 | 574,000 | 1,741,000 | 1,999,000 | 2,289,000 | 1,801,000 | 1,113,000 | 2,757,000 | 2,276,000 | 1,110,000 |
| 65–74 | 769,000 | 2,080,000 | 2,440,000 | 2,846,000 | 2,646,000 | 2,796,000 | 3,987,000 | 2,854,000 | 2,175,000 |
| ≥ 75 | 2,052,000 | 1,251,000 | 1,762,000 | 2,373,000 | 2,120,000 | 1,895,000 | 2,618,000 | 2,147,000 | 2,084,000 |
| Total[¶] | 5,516,000 | 7,877,000 | 10,580,000 | 11,493,000 | 9,929,000 | 9,997,000 | 14,199,000 | 10,355,000 | 7,997,000 |

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[*] All relative standard errors are < 30%, unless otherwise indicated
[†] Relative standard error of the estimate is 30%–50%, the estimate is unreliable
[§] Relative standard error of the estimate exceeds 50%
[¶] Numbers for each variable might not add to total because of rounding error and missing race data for 1990

Table 8. Estimated Annual Rate[*] of Physician Office Visits (1980–1998) and Hospital Outpatient Visits (1995–1998) for Chronic Obstructive Pulmonary Disease as the First-Listed Diagnosis, by Race, Sex, and Age Group—United States, National Ambulatory Medical Care Survey, 1980–2000, and National Hospital Ambulatory Medical Care Survey, 1995–2000[†]

| Variable | 1980 | 1985 | 1990 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|---|---|---|---|---|---|---|---|---|---|
| Race[§] | | | | | | | | | |
| White | 42.5 | 58.1[¶] | 67.1[¶] | 74.6[¶] | 60.2 | 63.7[¶] | 90.2[¶] | 61.8 | 46.9 |
| Black | 36.5[**] | 25.7[¶][**] | 31.2[¶][**] | 25.4[¶][**] | 49.3[**] | 32.8[¶][**] | 34.7[¶][**] | 55.6 | 30.4[**] |
| Other | —[††] | —[††] | 176.4[**] | 58.9[**] | 49.5[**] | —[††] | —[††] | —[††] | —[††] |
| Sex[§] | | | | | | | | | |
| Male | 45.7 | 57.4 | 65.3 | 74.2 | 60.6 | 62.5 | 78.7 | 51.9 | 46.8 |
| Female | 37.8 | 51.4 | 68.6 | 63.4 | 56.7 | 54.4 | 84.5 | 66.2 | 43.4 |
| Age group (y) | | | | | | | | | |
| 25–44 | 23.3 | 27.0 | 38.6 | 32.6 | 27.2 | 32.0 | 39.5 | 21.8 | 17.7 |
| 45–54 | 28.9 | 36.3 | 50.7 | 43.0 | 35.6 | 47.5 | 46.6 | 36.1 | 31.9 |
| 55–64 | 26.4 | 81.3 | 94.7 | 101.6 | 78.9 | 48.2 | 117.8 | 96.1 | 46.3 |
| 65–74 | 49.4 | 123.7 | 135.2 | 157.3 | 146.2 | 154.4 | 220.0 | 157.4 | 119.9 |
| ≥ 75 | 205.8 | 108.9 | 135.6 | 160.4 | 140.0 | 122.3 | 165.0 | 132.4 | 125.7 |
| Total[§] | 44.5 | 53.8 | 67.6 | 68.7 | 58.6 | 58.3 | 81.6 | 58.9 | 45.0 |

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[*] Per 1,000 population
[†] All relative standard errors are < 30%, unless otherwise indicated
[§] Age-adjusted to 2000 U.S. population
[¶] Represents a statistically significant difference between blacks and whites or males and females for that year
[**] Relative standard error of the estimate is 30%–50%, the estimate is unreliable
[††] Relative standard error of the estimate exceeds 50%

for COPD decreased, but during 1990–1999, hospitalization rates increased (Figs. 6 and 7). Hospitalization rates for COPD among whites were greater than those among blacks during 1980–1987, after which rates have been similar (Fig. 7). However, approximately ≤ 20% of hospitalizations did not have race listed.[22] Hospitalization rates for men were greater than those for women through the 1980s; however, since 1995 these rates have been similar (Fig. 6). Since 1990, hospitalizations for COPD have increased among all age groups, with the largest increases observed for those persons aged 65–74 years (62%) and those aged ≥ 75 years (52%).

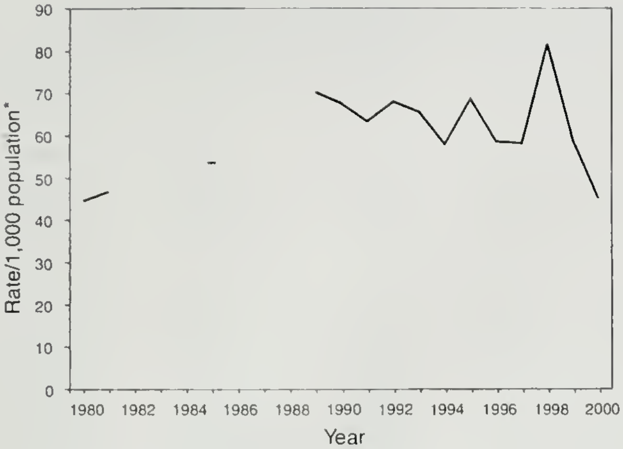

Fig. 4. Estimated annual rate of office visits with chronic obstructive pulmonary disease (COPD) as the first-listed diagnosis, by year, United States, from the National Ambulatory Medical Care Survey, 1980–1998, and National Hospital Ambulatory Medical Care Survey, 1992–2000. * Age-adjusted to 2000 U.S. population.

## Deaths

During 1980–2000, the overall death rate for COPD increased 67% (Tables 13 and 14). During this period, rates among whites increased 67%, and rates among blacks increased 87%. However, deaths rates for whites remained higher than those for blacks throughout the 21-year period. During 1980–2000, death rates for COPD among men increased 13%; however, death rates among men remained steady since 1985. During 1980–2000, death rates for COPD among women approximately tripled and increased steadily throughout the period (Fig. 8). During 2000, the number of women who died from COPD was, for the first time, higher than the number of men who died from COPD.

During 1999–2000, death rates for COPD were higher, compared with 1998. Because the comparability ratio for coding COPD under ICD-10 compared with ICD-9 is > 1, which results in a higher number of COPD deaths under ICD-10, increases observed for 1999 reflect in part the impact of a new disease classification system.

## Discussion

These data identified certain trends in COPD-related morbidity and mortality in the U.S. First, COPD-related deaths among women continued to increase. By 2000, the number of COPD deaths among women surpassed the number among men, although the population-based mortality rates remain higher among men. Second, since 1989, COPD hospitalizations have also increased, combined with an elimination of the difference in hospitalization rates between men and women. Third, the proportion of the population aged < 55 years with mild or moderate COPD, on the basis of PFT, decreased from 1971–1975 to 1988–1994, whereas this proportion did not change substantially among other age groups.

The increasing trends in COPD hospitalizations and mortality among women reported here probably reflect the increase in smoking by women since the 1940s, relative to men, in the U.S.[27] The natural history of COPD among smokers is that smoking behaviors start during youth, lung function decline becomes apparent when smokers reach age 40–50

Table 9. Estimated Annual Number of Emergency Department Visits for Chronic Obstructive Pulmonary Disease as the First-Listed Diagnosis, by Race, Sex, and Age Group—United States, National Hospital Ambulatory Medical Care Survey, 1992–2000*

| Variable | 1992 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|---|---|---|---|---|---|---|---|
| Race | | | | | | | |
| White | 913,000 | 1,128,000 | 930,000 | 1,084,000 | 1,114,000 | 1,205,000 | 1,278,000 |
| Black | 158,000 | 241,000 | 247,000 | 241,000 | 276,000 | 285,000 | 243,000 |
| Other | —† | 54,000§ | 58,000§ | —† | —† | —† | —† |
| Sex | | | | | | | |
| Male | 422,000 | 686,000 | 546,000 | 570,000 | 573,000 | 730,000 | 651,000 |
| Female | 673,000 | 738,000 | 689,000 | 766,000 | 863,000 | 802,000 | 898,000 |
| Age group (y) | | | | | | | |
| 25–44 | 366,000 | 503,000 | 457,000 | 486,000 | 517,000 | 448,000 | 481,000 |
| 45–54 | 114,000 | 180,000 | 180,000 | 134,000 | 201,000 | 270,000 | 194,000 |
| 55–64 | 136,000 | 228,000 | 140,000 | 210,000 | 187,000 | 269,000 | 315,000 |
| 65–74 | 255,000 | 243,000 | 262,000 | 227,000 | 263,000 | 233,000 | 267,000 |
| ≥ 75 | 224,000 | 269,000 | 196,000 | 280,000 | 268,000 | 312,000 | 292,000 |
| Total¶ | 1,095,000 | 1,423,000 | 1,235,000 | 1,336,000 | 1,436,000 | 1,532,000 | 1,549,000 |

*All relative standard errors are < 30%, unless otherwise indicated

†Relative standard error of the estimate exceeds 50%

§Relative standard error of the estimate is 30%–50%, the estimate is unreliable

¶Numbers for each variable might not add up to total because of rounding error

Table 10. Estimated Annual Rate* of Emergency Department Visits With Chronic Obstructive Pulmonary Disease as the First-Listed Diagnosis, by Race, Sex, and Age Group—United States, National Hospital Ambulatory Medical Care Survey, 1992–2000†

| Variable | 1992 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|---|---|---|---|---|---|---|---|
| Race§ | | | | | | | |
| White | 64.9¶ | 78.1¶ | 63.7¶ | 73.4¶ | 74.9¶ | 80.2¶ | 84.3¶ |
| Black | 88.7¶ | 142.8¶ | 134.6¶ | 126.5¶ | 147.8¶ | 156.7¶ | 130.8¶ |
| Other | —** | 91.6†† | 94.3†† | —** | —** | —** | —** |
| Sex§ | | | | | | | |
| Male | 57.5 | 90.0 | 70.8 | 74.1 | 72.7 | 93.0 | 80.7 |
| Female | 76.6 | 82.0 | 75.9 | 82.7 | 93.1 | 85.7 | 94.4 |
| Age group (y) | | | | | | | |
| 25–44 | 45.3 | 61.9 | 56.2 | 59.6 | 63.4 | 54.8 | 58.7 |
| 45–54 | 41.5 | 58.0 | 55.8 | 40.0 | 58.1 | 75.3 | 52.4 |
| 55–64 | 63.0 | 101.2 | 61.3 | 90.7 | 79.8 | 113.7 | 131.6 |
| 65–74 | 141.0 | 134.6 | 145.0 | 125.2 | 145.0 | 128.5 | 147.1 |
| ≥ 75 | 163.1 | 181.6 | 129.3 | 180.4 | 168.8 | 192.5 | 176.1 |
| Total§ | 67.6 | 84.9 | 72.7 | 77.6 | 82.6 | 87.4 | 87.2 |

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*Per 10,000 population.

†All relative standard errors are < 30%, unless otherwise indicated

§Age-adjusted to 2000 U.S. population

¶Represents a statistically significant difference between blacks and whites or males and females for that year

**Relative standard error of the estimate exceeds 50%.

††Relative standard error of the estimate is 30%–50%; the estimate is unreliable.

years, hospitalizations begin when smokers reach age 50–69, and deaths occur when they reach age 60–79.[1] The data in this report support this natural history with regard to different trends among men and women: that is, since the late 1980s women have had a similar hospitalization rate for COPD (Fig. 6), and since 1980 the COPD mortality gap between women and men has narrowed. However, the decreasing proportion of adults aged 25–54 years with objective evidence of COPD (Table 4) is most likely related to lower smoking prevalence among the U.S. population since the 1960s.[27]

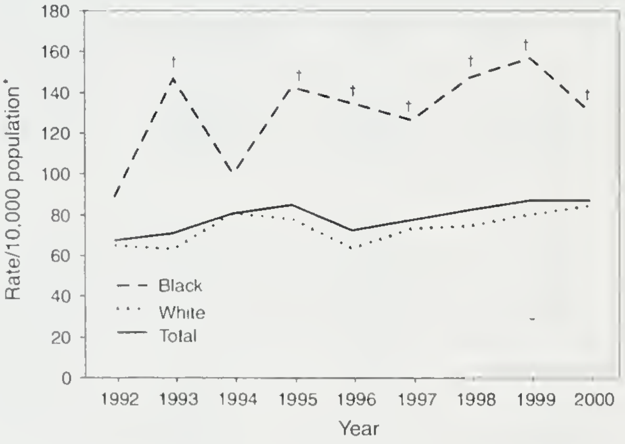

Fig. 5. Estimated annual rate of emergency department visits for chronic obstructive pulmonary disease (COPD) as the first listed-diagnosis, by race and year, United States, from the National Hospital Ambulatory Medical Care Survey, 1992–2000. * Age-adjusted to 2000 U.S. population. † Represents a statistically significant difference from rate among whites.

Objective measures are critical in determining whether COPD is present. Historically, COPD has been defined by the presence of certain symptoms (eg, cough and sputum production). Compared with COPD defined by using PFT, this historical method results in underdiagnosis of this condition, chiefly among older populations and among persons with mild disease.[3] The GOLD criteria[9] allow clinicians to diagnose and classify COPD on the basis of PFT. These criteria recognize that symptoms might be present or absent, even among persons with substantial degrees of impairment. For example, the data demonstrate that a higher proportion of men had evidence of obstructive lung disease than women (Table 4), which is supported by the consistent finding of a higher COPD death rate among men (Table 14). This can be contrasted with the higher proportion of women reporting COPD on the basis of physician diagnosis, which is not necessarily based on objective criteria.

During 1994–1996, approximately 8.0% of adults with COPD reported activity limitation caused by COPD (Table 5). Whether this measure captured completely the degree of disability associated with COPD is unclear because participants might not have accurately classified the cause of their disabilities or they might have comorbid conditions that cause disability also. For example, during 1994–1996, a total of 38.6% of participants with COPD and 18.4% of those without COPD reported activity limitation,

Table 11. Estimated Annual Number of Hospitalizations for Chronic Obstructive Pulmonary Disease, Stratified by Race, Sex, and Age Group—United States, National Hospital Discharge Survey, 1980–2000*

| Variable | 1980 | 1985 | 1990 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|---|---|---|---|---|---|---|---|---|---|
| Race | | | | | | | | | |
| White | 542,000 | 510,000 | 370,000 | 474,000 | 453,000 | 502,000 | 529,000 | 556,000 | 491,000 |
| Black | 35,000 | 36,000 | 31,000 | 63,000 | 57,000 | 56,000 | 61,000 | 68,000 | 58,000 |
| Other | 6,000† | 11,000 | 4,000† | 12,000 | 13,000 | 13,000 | 21,000 | 15,000 | 12,000 |
| Unidentified | 68,000 | 45,000 | 58,000 | 96,000 | 123,000 | 124,000 | 126,000 | 148,000 | 165,000 |
| Sex | | | | | | | | | |
| Male | 367,000 | 316,000 | 221,000 | 283,000 | 289,000 | 296,000 | 321,000 | 332,000 | 322,000 |
| Female | 285,000 | 286,000 | 242,000 | 362,000 | 358,000 | 400,000 | 416,000 | 455,000 | 404,000 |
| Age group (y) | | | | | | | | | |
| 25–44 | 60,000 | 40,000 | 34,000 | 41,000 | 35,000 | 40,000 | 37,000 | 41,000 | 37,000 |
| 45–54 | 71,000 | 53,000 | 44,000 | 72,000 | 65,000 | 74,000 | 63,000 | 69,000 | 77,000 |
| 55–64 | 154,000 | 142,000 | 87,000 | 109,000 | 121,000 | 118,000 | 137,000 | 144,000 | 134,000 |
| 65–74 | 204,000 | 204,000 | 143,000 | 210,000 | 196,000 | 205,000 | 227,000 | 236,000 | 202,000 |
| ≥ 75 | 163,000 | 164,000 | 156,000 | 213,000 | 230,000 | 259,000 | 273,000 | 298,000 | 276,000 |
| Total§ | 652,000 | 603,000 | 463,000 | 645,000 | 647,000 | 696,000 | 737,000 | 787,000 | 726,000 |

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*All relative standard errors are < 30%, unless otherwise indicated.

†Relative standard error of the estimate is 30%–50%; the estimate is unreliable.

§Numbers for each variable might not add to total because of rounding error

Table 12. Estimated Annual Rates* of Hospitalization for Chronic Obstructive Pulmonary Disease, Stratified by Race, Sex, and Age Group—United States, National Hospital Discharge Survey, 1980–2000†

| Variable | 1980 | 1985 | 1990 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|---|---|---|---|---|---|---|---|---|---|
| Race§¶ | | | | | | | | | |
| White | 45.3** | 39.6** | 27.2** | 32.4 | 30.6 | 33.5 | 34.8 | 36.1 | 31.5 |
| Black | 30.6** | 27.9** | 22.6** | 41.5 | 37.8 | 36.3 | 39.1 | 43.1 | 36.0 |
| Other | 39.9†† | 39.5 | 13.0†† | 26.3 | 28.2 | 27.9 | 43.4 | 28.3 | 21.3 |
| Sex§ | | | | | | | | | |
| Male | 65.3** | 52.3** | 35.1** | 40.2 | 40.9 | 41.0 | 43.9 | 44.4 | 42.4 |
| Female | 38.2** | 35.1** | 28.0** | 38.6 | 37.5 | 41.5 | 42.5 | 45.7 | 40.2 |
| Age group (y) | | | | | | | | | |
| 25–44 | 9.5 | 5.6 | 4.2 | 5.1 | 4.4 | 4.9 | 4.5 | 5.0 | 4.5 |
| 45–54 | 31.2 | 22.3 | 17.4 | 23.2 | 20.1 | 22.1 | 18.1 | 19.1 | 20.8 |
| 55–64 | 71.1 | 66.1 | 41.4 | 48.2 | 52.9 | 51.2 | 58.8 | 67.4 | 55.9 |
| 65–74 | 131.0 | 121.6 | 79.1 | 115.9 | 108.4 | 113.4 | 125.2 | 130.1 | 111.6 |
| ≥ 75 | 163.0 | 142.5 | 120.0 | 144.2 | 151.7 | 160.9 | 172.0 | 184.0 | 166.3 |
| Total§ | 49.0 | 41.8 | 30.4 | 38.9 | 38.5 | 40.9 | 42.6 | 44.9 | 40.8 |

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*Per 10,000 population

†All relative standard errors are < 30%, unless otherwise indicated

§Age-adjusted to 2000 U.S. population.

¶Rates by race are underestimates because of the substantial proportion of unidentified race data (see Table 11).

**Represents a statistically significant difference between blacks and whites or males and females for that year

††Relative standard error of the estimate is 30%–50%; the estimate is unreliable.

indicating that COPD-associated activity limitation might be higher than the 8.0% reported or that other comorbid diseases, which are possibly smoking-related, occur more frequently among persons with COPD. The data regarding reported functional limitation indicated a higher prevalence of limitation among participants with COPD (Table 6). Actual testing of functional capabilities relevant to COPD (eg, a 6-min walk) could not be performed in NHANES III because of time limitations.

The dramatic drop in COPD hospitalizations during 1983–1989 is probably related to systematic changes in U.S. health care at that time (eg, introduction of diagnosis-

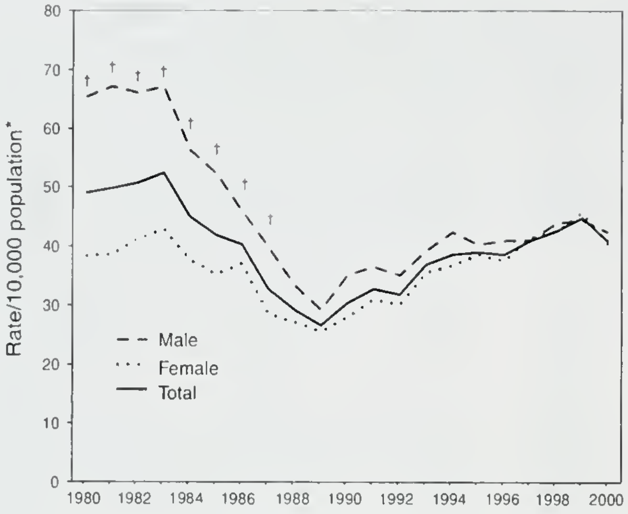

Fig. 6. Estimated annual rate of hospitalizations with chronic obstructive pulmonary disease (COPD) as the first-listed diagnosis by sex and year, United States, from the National Hospital Discharge Survey, 1980–2000. * Age-adjusted to 2000 U.S. population. † Represents a statistically significant difference from rate among females.

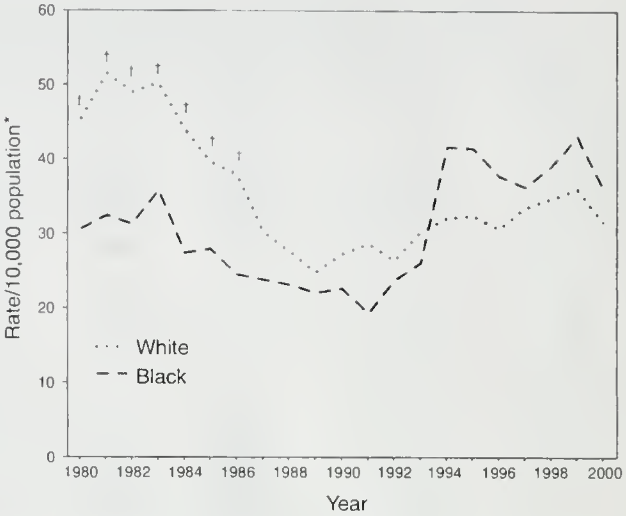

Fig. 7. Estimated annual rate of hospitalizations with chronic obstructive pulmonary disease (COPD) as the first-listed diagnosis, by race and year, United States, from the National Hospital Discharge Survey, 1980–2000. * Age-adjusted to 2000 U.S. population. † Represents a statistically significant difference from rate among blacks.

related groups for compensation, overall pressures within the health care system to decrease hospitalizations, or other unknown factors). Our observation that COPD mortality steadily increased throughout this period would indicate that the observed decrease was not related to a lower prevalence of severe COPD among the population. Since 1989, COPD hospitalization rates have steadily increased, with the rate among women now similar to that among men, and the rate among blacks similar to that among whites.

Tobacco use is the key risk factor in COPD development and progression, and trends in COPD mortality among women reported here reflect the recent increase in smoking by women, relative to men, in the U.S.[27] Although tobacco smoking is the most critical risk factor for both development and progression of COPD, asthma,[28] exposure to ambient pollutants in the home and workplace,[29] and respiratory infections[30,31] are also key factors. In ad-

Table 13. Annual Number of Deaths With Chronic Obstructive Pulmonary Disease as the Underlying Cause of Death, Stratified by Race, Sex, and Age Group—United States, Mortality Component of the National Vital Statistics System, 1980–2000*

| Variable | 1980† | 1985 | 1990 | 1995 | 1996 | 1997 | 1998 | 1999§ | 2000 |
|---|---|---|---|---|---|---|---|---|---|
| Race | | | | | | | | | |
| White | 49,223 | 65,445 | 75,391 | 89,605 | 92,445 | 95,415 | 98,691 | 110,165 | 111,260 |
| Black | 2,735 | 3,802 | 4,586 | 5,329 | 5,501 | 5,614 | 5,830 | 6,679 | 6,379 |
| Other | 235 | 467 | 679 | 927 | 934 | 1,071 | 1,069 | 1,295 | 1,415 |
| Sex | | | | | | | | | |
| Male | 36,878 | 44,598 | 47,053 | 51,349 | 51,925 | 53,477 | 54,466 | 60,311 | 59,118 |
| Female | 15,315 | 25,116 | 33,603 | 44,512 | 46,955 | 48,623 | 51,124 | 57,828 | 59,936 |
| Age group (y) | | | | | | | | | |
| 25–44 | 302 | 286 | 314 | 387 | 399 | 439 | 421 | 494 | 506 |
| 45–54 | 1,869 | 1,868 | 1,748 | 2,057 | 2,106 | 2,171 | 2,151 | 2,472 | 2,627 |
| 55–64 | 8,616 | 9,741 | 9,506 | 9,123 | 9,133 | 9,283 | 9,410 | 10,643 | 10,338 |
| 65–74 | 19,127 | 23,986 | 26,155 | 28,710 | 28,837 | 29,332 | 29,879 | 31,699 | 31,041 |
| ≥ 75 | 22,279 | 35,058 | 42,933 | 55,584 | 58,405 | 60,875 | 63,729 | 72,831 | 74,542 |
| Total | 52,193 | 69,714 | 80,656 | 95,861 | 98,880 | 102,100 | 105,590 | 118,139 | 119,054 |

*All relative standard errors are < 30%.

†Codes 490–492 (1980–1998) from World Health Organization. Manual of the international statistical classification of diseases, injuries, and causes of death, 9th revision. Geneva: World Health Organization, 1977.

§Codes J40–J44 (1999–2000) from World Health Organization. International statistical classification of diseases and related health problems, 10th revision. Geneva: World Health Organization, 1992.

Table 14. Annual Rates* for Deaths With Chronic Obstructive Pulmonary Disease as the Underlying Cause of Death, Stratified by Race, Sex, and Age Group—United States, Mortality Component of the National Vital Statistics System, 1980–2000†

| Variable | 1980§ | 1985 | 1990 | 1995 | 1996 | 1997 | 1998 | 1999¶ | 2000 |
|---|---|---|---|---|---|---|---|---|---|
| Race | | | | | | | | | |
| White** | 42.4†† | 52.0†† | 55.4†† | 60.9†† | 61.9†† | 63.0†† | 64.2†† | 70.6†† | 70.1†† |
| Black | 24.6†† | 32.3†† | 36.4†† | 38.9†† | 39.6†† | 39.7†† | 40.6†† | 46.0†† | 42.9†† |
| Other | 16.3 | 22.3 | 25.7 | 25.3 | 24.6 | 26.6 | 25.0 | 29.1 | 30.6 |
| Sex** | | | | | | | | | |
| Male | 73.0†† | 81.9†† | 80.0†† | 78.9†† | 78.3†† | 79.0†† | 79.0†† | 85.9†† | 82.6†† |
| Female | 20.1†† | 30.2†† | 37.0†† | 45.4†† | 47.2†† | 48.1†† | 49.9†† | 55.6†† | 56.7†† |
| Age group (y) | | | | | | | | | |
| 25–44 | 0.5 | 0.4 | 0.4 | 0.5 | 0.5 | 0.5 | 0.5 | 0.6 | 0.6 |
| 45–54 | 8.2 | 7.8 | 7.0 | 6.6 | 6.5 | 6.5 | 6.2 | 6.9 | 7.1 |
| 55–64 | 39.7 | 45.5 | 45.0 | 40.5 | 40.0 | 40.2 | 40.2 | 45.0 | 43.1 |
| 65–74 | 122.8 | 142.7 | 144.9 | 158.7 | 159.3 | 162.0 | 164.9 | 174.9 | 171.2 |
| ≥ 75 | 223.5 | 294.6 | 330.3 | 375.9 | 385.7 | 392.7 | 401.8 | 449.1 | 449.7 |
| Total** | 40.7 | 50.0 | 53.3 | 58.4 | 59.3 | 60.2 | 61.3 | 67.6 | 66.9 |

*Per 100,000 population
†All relative standard errors are < 30%.
§Codes 490–492 and 496 (1980–1998) from World Health Organization Manual of the international statistical classification of diseases, injuries, and causes of death, 9th revision. Geneva: World Health Organization, 1977
¶Codes J40–J44 (1999–2000) from World Health Organization. International statistical classification of diseases and related health problems, 10th revision Geneva World Health Organization, 1992
**Age-adjusted to 2000 U.S population
††Represents a statistically significant difference between blacks and whites or males and females for that year

dition, COPD is a risk factor for other outcomes (eg, lung cancer or early death).[32,33] Early detection of COPD might alter its course and prognosis. The National Lung Health Education Program (NLHEP) is a new health care initiative aimed at involving primary-care physicians in early identification and treatment of obstructive lung disease.[34] NLHEP is promoting widespread use of simple office spirometry to measure pulmonary function among current and former smokers aged ≥ 45 years and anyone with respiratory symptoms. Although spirometry is a relatively easy test to perform, it does require training of both the test administrator and the patient to obtain an accurate result.

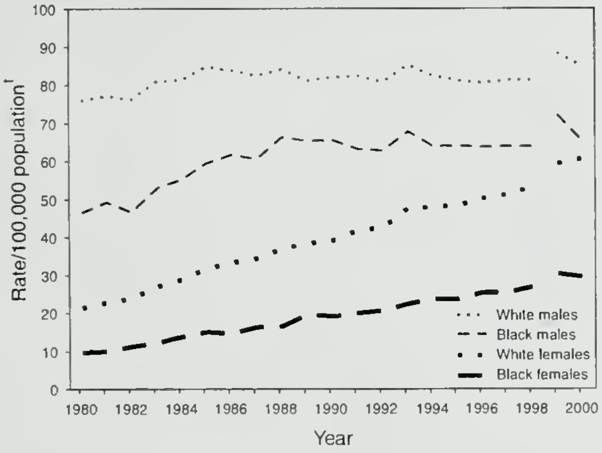

Fig. 8. Annual rate of death from chronic obstructive pulmonary disease (COPD) as the underlying cause of death (Codes 490–492 and 496 [1980–1998] from the World Health Organization manual of the international statistical classification of diseases, injuries, and causes of death, 9th revision. Geneva: World Health Organization, 1977), by sex, race, and year, United States, from the Mortality Component of the National Vital Statistics System, 1980–2000. † Codes J40-J44 (1999–2000) from the World Health Organization international statistical classification of diseases and related health problems, 10th revision. Geneva: World Health Organization, 1992.

Two *Healthy People 2010* objectives relate to COPD: reduce the proportion of adults whose activity is limited because of chronic lung and breathing problems among adults aged ≥ 45 years to 1.5% in 2010 (Objective 24–9), compared with 2.2% in 1997, and reduce deaths from COPD among adults aged ≥ 45 years to 60 deaths/100,000 in 2010, compared with 119.4 deaths/100,000 in 1998 (Object 24–10)[5] (the 1998 estimate of 61.3 deaths/100,000 in this report is for adults aged ≥ 25 years, and a 50% decrease would result in a goal of 30 deaths/100,000). The first goal, which is related both to the prevalence of COPD among adults and the proportion of those with activity limitation, seems achievable, although, as noted previously, persons with COPD might underestimate the amount of their activity limitation that is related to COPD. The second goal, given the continued high rates of COPD mortality in 1999 and 2000 and the aging of the population, will likely not be achieved if the current trends continue.

The greatest challenge in COPD surveillance is related to the case definition for COPD. If one assumes that persons with moderate obstructive lung disease have clinically significant COPD, using chronic bronchitis in addition to emphysema to define COPD in surveys probably overestimates COPD among younger populations and underestimates it among older ones (Fig. 1). Although the term COPD is increasingly being used by both patients and physicians, it is not typically used in health surveys. In addition, depending on physician diagnosis is problematic because COPD is frequently silent and, in the absence of spirometry, often undiagnosed.[3] Even using spirometry, as in NHANES I and III, might result in the misclassification of conditions among certain persons who have short-term reversibility in their airway obstruction, because reversibility testing has not been done in national surveys in the U.S. Finally, although we did not include asthma in this report, asthma and COPD can coexist and can be difficult to differentiate, chiefly among older populations.[3]

The continuing increase in COPD hospitalizations among men and women and the increase in COPD deaths among women is problematic and highlights the need for both clinical and public health interventions. Conversely, the decrease in COPD, as determined by PFT, among persons aged < 55 years, might indicate that tobacco-control efforts and other efforts to improve respiratory protection in recent years might be having a positive effect that will ultimately result in less COPD morbidity and mortality.

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# Letters

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## Evaluating a New Blood Gas Sensor

Evaluating a new measuring device that promises benefits over an older device poses some interesting problems. We can compare measurements made with the new device to known values and determine its accuracy. Alternatively, we can compare the new device to an older device whose accuracy is assumed and determine if the new one will be a suitable replacement. The study designs and data analyses are similar in both cases. However, there are important underlying philosophical issues that are often confused. As a result, there is a tendency for authors to "go through the motions" of acceptable statistical analysis while missing the point in their conclusions. A recent article in RESPIRATORY CARE illustrates this.[1]

Meyers et al evaluated a continuous intravascular blood gas sensor (Neotrend, Diametrics Medical, Palo Alto, California) to determine if it "... would produce clinically acceptable bias and precision in comparison to laboratory values.... "[1] Sensor measurements were compared with arterial blood gas measurements made with a standard laboratory analyzer. The differences between sensor and laboratory values were used to calculate bias and precision, as suggested by Bland and Altman.[2] The authors then concluded that the device was "accurate."

The study is well done and the authors are to be congratulated for conducting a useful study and using the appropriate data analysis. But there are 2 problems with their conclusion. First, studies of accuracy, by definition, require that measured values be compared to some type of accepted standard values.[3–6] The accepted standard for blood gas measurement is whole blood, tonometered to specific values, using precision gas mixtures. In contrast, Meyers et al compared measured values from the intravascular sensor with values from a standard laboratory analyzer. The values from the laboratory analyzer are, strictly speaking, not the true values, because we know there are measurement errors. That is why the Bland-Altman plots of the data show the mean value of each data pair along the horizontal axis; we don't know which one of the pair is the true value, so our best guess is the average of the two.[2] The method used in the Meyers et al study is appropriate for the evaluation of *agreement between 2 methods*, not for determining the *accuracy of a new device.* Bland and Altman never even mentioned the terms *accuracy* and *precision.*[2]

The idea behind evaluating agreement is to estimate the systematic error by calculating the mean difference between the new device and the conventional device. Then the random error is expressed as the standard deviation of the differences. The resultant total error or "limits of agreement" are then calculated as the mean difference ± 2 standard deviations. In other words, 95% of all future measurements with the new device should agree with the standard device by being within those limits. If the limits are small enough, we may conclude that the new device can be used in place of the standard device, with no effect on the quality of care.

Though my first objection to the study's conclusion may be splitting hairs, the second is more problematic because it could adversely affect patient outcomes. Meyers et al set out to determine if the intravascular blood gas sensor would produce "clinically acceptable" results. However, they never defined what clinically acceptable means. How large would the limits of agreement have to be to reject the device as clinically unacceptable?

As explained in detail elsewhere,[5] acceptable differences between new and conventional measurements must be established a priori. These standards can be derived in a number of ways, but essentially one must decide if the expected difference will be of clinical importance (ie, that the clinical decision might differ depending on which instrument was used for the measurement). For example, in our blood gas laboratory the maximum acceptable difference in $P_{O_2}$ values on a split sample measured on 2 similar machines is 7 mm Hg. Thus, it would be reasonable for our lab to demand that any new device provide individual measurements whose limits of agreement are at most ± 7 mm Hg, to preserve our standard of care. This type of reasoning has been used in other studies of similar devices.[7]

Meyers et al found that the worst-case expected difference between the intravascular sensor and the laboratory values (ie, bias minus 2 standard deviations) was as follows:

pH: −0.06
$P_{aCO_2}$: −11 mm Hg
$P_{aO_2}$: −31 mm Hg

with the negative sign indicating that the sensor value is larger than the laboratory value (because the limits were calculated as laboratory value minus sensor value).

The question that Meyers et al should have addressed in their conclusion is whether such differences are clinically acceptable. They could have simply said that, in their opinion, differences between laboratory and sensor values of 0.06 units for pH, 11 mm Hg for $P_{aCO_2}$, and 31 mm Hg for $P_{aO_2}$ are acceptable, and anything higher is not. They did not do that. Instead they stated that "... data from the Neotrend sensor fall within the accuracy range required for discrete blood gas analyzers." This statement is unexplained, unreferenced, and, in my opinion, misleading. As I suggested earlier, split sample results run on identical blood gas analyzers show less difference. Section 493.1213(b) of the Clinical Laboratory Improvement Amendments of 1988 (CLIA) standards states: "Although no specific guidelines exist for verifying a test method, each laboratory is responsible for determining the performance characteristics of its own methods... verification may be accomplished by comparison of split sample results with results obtained from a method that has been shown to provide clinically valid results." Since the College of American Pathologists guidelines for blood gas laboratories recommend split sample testing among similar analyzers as an ongoing quality control measure, laboratories approved by the College of American Pathologists should already have the type of agreement data necessary to make such comparisons. These are the criteria each lab should use in determining whether to accept new technology. Proficiency testing standards mentioned in CLIA are not really appropriate, because they were meant for comparing a laboratory's performance with 10 or more refereed laboratories. There are more sources of error between laboratories

Table 1. Simulated Results of Split Sample Comparisons of Laboratory Values Versus Intravascular Sensor Values

| | Physiologic Ranges | | | | | |
|---|---|---|---|---|---|---|
| | pH | | $P_{CO_2}$ | | $P_{O_2}$ | |
| | Low | Hi | Low | Hi | Low | Hi |
| Possible range | 7.20 | 7.50 | 40 | 60 | 40 | 90 |
| Target range | 7.25 | 7.45 | 45 | 55 | 50 | 80 |

| Difference (lab value minus sensor value) | | | | | |
|---|---|---|---|---|---|
| pH | | $P_{CO_2}$ | | $P_{O_2}$ | |
| Bias | Precision | Bias | Precision | Bias | Precision |
| 0.00 | 0.03 | −3 | 4 | 4 | 13.5 |

| Sample | Lab Values | | | Ventilator Change | Sensor Values | | | Ventilator Change |
|---|---|---|---|---|---|---|---|---|
| | pH | $P_{CO_2}$ | $P_{O_2}$ | | pH | $P_{CO_2}$ | $P_{O_2}$ | |
| 1 | 7.47 | 55 | 64 | Yes | 7.51 | 60 | 82 | Yes |
| 2 | 7.41 | 55 | 75 | No | 7.46 | 53 | 67 | Yes |
| 3 | 7.40 | 49 | 74 | No | 7.44 | 45 | 70 | No |
| 4 | 7.47 | 46 | 65 | Yes | 7.43 | 44 | 45 | Yes |
| 5 | 7.46 | 44 | 51 | Yes | 7.42 | 44 | 46 | Yes |
| ... | ... | ... | ... | ... | ... | ... | ... | ... |
| 3000 | 7.34 | 48 | 70 | No | 7.31 | 52 | 92 | Yes |
| | | | % Yes | 79% | | | % Yes | 86% |

than within a single laboratory, so those criteria would be too loose for assessing the acceptability of a new device introduced to the practice of one hospital.

What happens if we accept less stringent standards? This is a question that is usually ignored in device evaluation studies. Most authors simply assert that the new device is or is not acceptable based on some arbitrary criteria and never consider the subsequent effect on clinical decisions. Keep in mind that the subjects in the Meyers et al study were neonates with respiratory failure, presumably on mechanical ventilation. Therefore, we might frame the question in terms of whether a clinical decision, such as a ventilator setting change, might be indicated simply as a result of the type of blood gas analyzer used, as opposed to a real clinical condition. In other words, would using the intravascular sensor in place of a laboratory analyzer cause any important change in the standard of care?

For example, a $P_{aCO_2}$ of $> 50$ mm Hg might be a clinical decision point indicating the need to increase minute ventilation, whereas a $P_{aCO_2}$ of 35–45 mm Hg might indicate that no change was needed. Suppose the patient's true $P_{aCO_2}$ was 41 mm Hg. Using the limits of agreement found in the Meyers et al study, it would be possible for the laboratory analyzer to give a reading of 40 mm Hg while the intravascular sensor gave a reading of 51 mm Hg. Thus, if we were relying on the intravascular sensor in place of the normal laboratory measurement, we would be inclined to make an unnecessary ventilator change. Such a change might be to increase the tidal volume and hence increase the risk of lung injury. The more unnecessary changes we make, the more risk the patient might incur and the longer the duration of ventilation might be.

Is this just an extreme example? Let's conduct a simulation to see. Suppose that we have guidelines for ventilating neonates with respiratory failure.[8] These guidelines provide a set of target ranges for pH, $P_{aCO_2}$, and $P_{aO_2}$. Measured values outside of the target ranges result in a change in the ventilator settings. A simulation is then created using a computerized spreadsheet program, such as Microsoft Excel, as follows:

1. *Generate simulated laboratory analyzer values.* Randomly select a value within some possible physiologic range for each blood gas variable. Because the current standard of care is based on laboratory values, we consider the random values to be the results of a standard blood gas analyzer.

2. *Generate simulated intravascular sensor values.* First we use the spreadsheet to simulate values for the difference between the intravascular sensor and laboratory readings by randomly selecting numbers from 3 normal distributions; 1 for pH, 1 for $P_{CO_2}$, and 1 for $P_{O_2}$, each with the mean and standard deviation values observed by Meyers et al. Then these differences are subtracted from the simulated lab values to get simulated sensor values (because, from the Meyers et al report, the difference was the laboratory value minus the sensor value).

3. *Evaluate the need for a ventilator change.* Finally, we compare the simulated lab values and sensor values to the target ranges for pH, $P_{aCO_2}$, and $P_{aO_2}$. If any variable is out of its target range, we make a ventilator change. Table 1 shows a portion of the data from 3,000 simulated blood gas determinations.

Given the assumptions I used for this simulation, the simulated blood gas laboratory values indicated ventilator changes 79% of the time, compared to 86% of the time based on the intravascular sensor readings. That difference is significant: $p < 0.0001$.

Repeated computer simulations give similar results. Thus it is reasonable to conclude that using the intravascular sensor in place of conventional laboratory analysis would result in unnecessary ventilator changes 5% of the time and thus change the standard of care. When you consider the number of ventilator changes made per month in a large nursery, 5% seems like a lot of wasted time. Whether those ventilator changes would affect patient outcomes is pure speculation, but we can say for sure that it would cost more in terms of labor hours per ventilator day.

Not surprisingly, as the differences between the laboratory values and sensor values become smaller (ie, smaller bias and precision for pH, $P_{aCO_2}$, and $P_{aO_2}$), the difference in clinical decisions becomes smaller. In fact, you can easily adjust the bias and precision values in the spreadsheet until the difference in percentage of *Yes* answers is acceptable. When bias and imprecision are set to zero, there is no longer any difference in the percentage of *Yes* answers. This may be a useful procedure for setting acceptable limits of agreement a priori. Though you still end up with an arbitrary threshold, at least it has a more obvious clinical relevance. The moral of the story is that, if there is any difference between old and new devices, there will always be some effect on clinical decision-making.

It is easy to be seduced by the convenience and speed of bedside measurement devices, especially when they offer other advantages such as reduced blood loss. But if we sacrifice scientifically based decisions for such assumed benefits, we may pay hidden costs in the long run. If we accept less precise blood gas measurements, our ventilator and drug therapy decisions will be less precise and hence might increase costs and length of stay and might affect outcomes. It seems to me that it would be a lot more practical to avoid this possibility by maintaining accuracy standards now than to try to prove in the future that reducing standards does no harm.

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*The authors respond:*

We read with interest and thank Mr Chatburn for his comments regarding our recent study.[1] The crux of the matter is his contention that the continuous measurement technique we describe may provide misleading information that could result in unnecessary or incorrect ventilator changes. He bases that conclusion on hypothetical examples of possible patient situations. The exercise is interesting, but we disagree with the conclusions drawn. He also comments on the analysis techniques used and discusses what "clinically acceptable" really means.

First we are taken to task for our use of the term "accurate," and Mr Chatburn suggests we "go through the motions of acceptable statistical analysis while missing the point." Clinical trials are difficult and time-consuming to perform. To suggest we would design a trial only to "go through the motions of acceptable statistical analysis" implies either that we did not know the correct analysis to perform or that we did not care. Neither are true. We used the Bland-Altman technique precisely because of its appropriateness for this type of study, as a number of other authors have done.[2–6] We used the term "accurate" in discussing our findings. We did not compare the sensor in vitro to tonometered gas specimens. We did, however, compare our measured values to a widely used and validated clinical laboratory analyzer that is generally accepted as producing "accurate" values, even though exact agreement between laboratory analyzers and tonometered specimens is neither found nor expected. As Mr Chatburn points out, we show agreement between the 2 methods as opposed to "accuracy" per se. Is this accurate enough? We believe the readers can evaluate our work, as well as that performed by others, and draw their own conclusion.

Regarding CLIA standards, we removed some of this information during the revision process. But in CLIA section 493.927 we find criteria for acceptable performance of $P_{O_2}$ = target value ± 3 mm Hg; $P_{CO_2}$ = target value ± 5 mm Hg; pH = target value ± 0.04 pH units.[7] We used the laboratory analyzer as the target value; our findings fall within the latter ranges.

We agree with Mr Chatburn regarding the need for caution when a new monitoring or measurement tool is introduced. Nowhere did we suggest that this new monitoring technique should replace those currently in use. In addition to the Neotrend monitor, we use continuous oxygen saturation monitoring, intermittent laboratory analysis of arterial blood gases, and continuous monitoring of other physiologic variables. When one indicator is out of line with either the clinical situation or another similar monitor, further testing and investigation is necessary. This does not mean that continuous information should be discarded because it may not exactly reflect values generated in the central laboratory. Mr Chatburn's arguments could also be used to suggest that transcutaneous techniques such as continuous oxygen saturation monitoring should be discarded. We believe that this technique, like all others used in patient management, require good clinical judgment in their application. When used appropriately, continuous monitoring has the potential to improve patient management.

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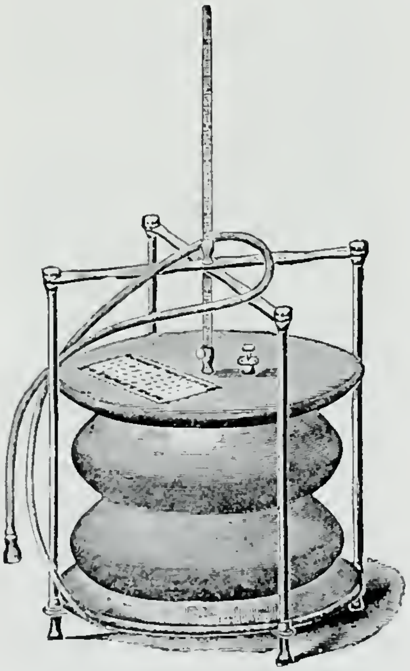

Denison's Spirometer from *Exercise and Food for Pulmonary Invalids*
Charles Denison AM MD Denver The Chain & Hardy Co 1895
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**High Altitude: An Exploration of Human Adaptation.** Thomas F Hornbein and Robert B Schoene, Editors. (Lung Biology in Health and Disease series, Volume 161, Claude Lenfant, Executive Editor.) New York: Marcel Dekker. 2001. Hard cover, illustrated, 982 pages, $235.

As a resident of moderate altitude I was eager to receive and read this latest edition of the Lung Biology in Health and Disease series. **High Altitude: An Exploration of Human Adaptation** is volume 161 in this ongoing series that deals with a wide range of topics relating to lung biology and disease. Surprisingly, it is the first volume that focuses on high altitude, and there are only 2 other volumes focusing on special environments (Volume 132, *The Lung at Depth*, and Volume 160, *Gravity and the Lung: Lessons from Microgravity*). However, the current volume goes beyond what has been done previously, as it not only addresses the lung at altitude but discusses multiple systems, all of which require adaptation for human survival at altitude. Owing to that ambitious goal, the size of the volume (an introduction plus 25 chapters, comprising 982 pages) and the large list of contributing authors (45) did not surprise me. I expected that reviewing this large volume would be a difficult task, but I didn't anticipate that on several occasions I would not be able to put it down.

In the preface the editors state that their goal was to begin to understand how low $P_{O_2}$ is sensed, translated, and leads to adaptation. To do this the chapters are grouped into 4 broad categories: (1) the stage, (2) organism defense, (3) oxygen's journey from air to mitochondrion, and (4) maladaptation. Each part encompasses 2 to 16 chapters. The overall organization is quite sound, with a logical progression from the history of our understanding of gases in the atmosphere (Chapters 1 and 2), to anthropologic evidence of long-term adaptation to residence at high altitude (Chapter 3), to sensing of oxygen and adaptation both in cells and organ systems (Chapters 6–21), to discussion of when adaptive mechanisms fail (Chapters 22–25).

The introductory chapters are very well written and compelling. Specifically, the first chapter, regarding the historical discovery of the nature of the air, is very engaging and instructive. The discussion of the atmosphere in Chapter 2 is equally compelling in detailing when and where humans have conquered high altitude. The anthropologic evidence for human adaptation to high altitude is equally compelling. However, the discussion of reproductive issues at high altitude should have been included as a separate chapter, as it highlights another organ system adaptation to high altitude.

The next section, which deals with oxygen sensing mechanisms and especially cellular defenses against hypoxia, is somewhat sparse and not completely up to date, perhaps owing to the complex mechanisms, which remain poorly understood. The attention paid to mechanisms of oxygen sensing in the carotid body is very appropriate and clearly written.

The next group of 21 chapters detail hypoxia adaptation with an organ-systems and physiologic response approach. I found this approach very effective, especially since it did not look exclusively at the lung and respiratory system but took a broader view. The inclusion of topics such as lung mechanics, neurobiologic behavior, renal function, immunologic responses, and circulatory responses to hypoxia make this a compelling summary of complex adaptation to hypoxia. Again, I would have included the reproductive system in this section and perhaps a discussion of aging and exposure to altitude as well. (This was also covered somewhat in Chapter 3.)

As in any collaborative text of this size, there is some repetition, but in this text the amount of repetition was not excessive and was appropriate given the probable use of this text. Several of the chapters (3, 9–13, 15–18, and 22–25) include summaries that highlight key unresolved issues and directions for future research. I found those summaries very helpful and thought the summary format should have been followed in more of the chapters, which would have made some of the longer chapters more useful as reference text.

The last 3 chapters deal with maladaptation to altitude, such as acute and chronic mountain sickness and high-altitude edema (cerebral and pulmonary). These chapters provide clear and concise information regarding clinical presentation, treatment, and prevention of these altitude-related diseases. I especially enjoyed the last chapter, which dealt with how altitude affects chronic illness, pregnancy, and alcohol consumption. These are the problems most likely to occur as more and more low-altitude residents venture to high altitude for work and pleasure. I am certain that these are the chapters I will refer to time and again.

**High Altitude: An Exploration of Human Adaptation** will have broad appeal to hypoxia researchers, physicians and other care providers, and mountaineers and other adventurists. The book may, however, have its biggest appeal to those interested in how humans adapt to various environments. The book's structure and attention to organ systems and physiologic responses makes it an easily-accessible information source. The figures and illustrations were adequate to explain key experiments and ideas, but, as in many books of this type, there could have been more; this is a difficult balance to find.

The subject index appears to be thorough and accurate, but the author index is cumbersome; the pages cited for an author refer to the place in the text where that author's work is cited, but the text cites the reference as a *number*, so to find an author you must scan the page for citation numbers, turn to that group of references, and then look for the author.

I found no typographical errors in the text or figures. Though the volume is well made and sturdy, because of its size and weight it is not easy to read away from a similarly sturdy desk.

Overall, I think that those interested in the complex manner in which humans adapt to altitude will find this book an excellent resource. As more of us venture higher and higher, understanding these complex physiologic processes and the failure of adaptation will become more critical.

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**Gravity and the Lung: Lessons from Microgravity.** G Kim Prisk, Manuel Paiva, and John B West, Editors. (Lung Biology in Health and Disease series, Volume 160, Claude Lenfant, Executive Editor.) New York: Marcel Dekker. 2001. Hard cover, illustrated, 383 pages, $165.

This book is Volume 160 in the National Heart, Lung, and Blood Institute's series on lung physiology and pathology in both normal and abnormal environments. The objective is to examine the influence of gravity (both increases and decreases thereof) on lung function, including lung volumes, chest wall mechanics, ventilation distribution, perfusion distribution, gas exchange, and pulmonary interstitial fluid balance. There are 15 contributors from 5 countries, all of whom are recognized leaders in their subjects. The quality of the text and authors is exemplified by the co-editors, Manuel Paiva, Kim Prisk, and above all, John West.

This text describes the effects of microgravity (ie, < 1 G, as occurs during space flight) on lung function. Though there is a moderate amount of scientific data directly addressing the effects of microgravity, the logistic limitations imposed by the circumstances have thus far limited the collection of direct data (earth-based models designed to simulate microgravity are imperfect at best). In addition to analyzing direct scientific data, the authors attempt to address voids by using information from earth-based models (when appropriate), parabolic flights (which can provide 22–27 s of microgravity), mathematical models, and extrapolation from experiments conducted in conditions exceeding 1 G (super-G forces). Most of the chapters (except Chapters 1, 2, and 14) begin by outlining the physiologic behavior of a specific aspect of lung function under normal gravitational force (1 G) and then use the above-mentioned sources of information to outline the effects of microgravity. The ultimate objective is to ascertain if the effects of microgravity on various aspects of lung function increase the probability of lung dysfunction in individuals undertaking prolonged space flight. That information might lead to mitigations of or solutions for that dysfunction.

This book is clearly not redundant in that there is no other repository of the detailed information contained in it. I suspect this book will be a necessity even for those who specialize in any one of the areas discussed, as they would benefit from the detailed discussions of the other areas. This book is also a worthy read for those who have a broader and general interest in pulmonary medicine, in that the detailed application of physiologic principles enunciated and the implications for diseases and their treatments represent the foundations of pulmonary medicine. For example, the chapter that discusses aerosol transport in the lung and the importance of particle size has clear implications for both the risk of developing airborne diseases and effective delivery of therapies.

The first chapter, written by John West, in and of itself justifies purchasing this book. It details important historical milestones in the field as far back as the late 19th century, and is fascinating reading that is wonderfully summarized and presented.

Although Chapter 2 is also a history chapter, it is very different from Chapter 1. It deals with the effects of higher gravity, and though the information presented is not of very recent vintage, the research discussed was conducted predominately in the latter half of the 20th century. This chapter outlines the effects of gravity on pulmonary variables, including mechanics, ventilation, perfusion, and ventilation-perfusion ratio, and the uses of these data to make predictions about microgravity (the errors and risks of such extrapolations are well discussed throughout the book; for example, regarding central venous pressure). Like the book as a whole, this is an information-rich chapter and is not casual reading.

Chapter 3 discusses the effects of gravity alterations on lung volumes and chest wall mechanics. Chapter 4 extends the discussion to ventilation distribution and includes an excellent discussion of ventilatory heterogeneity (described as *nonhomogeneity*), inter-regional versus intra-regional differences, and the relative contributions of gravitational versus nongravitational influences. Chapter 5 discusses aerosol delivery in the tracheobronchial tree. Chapters 6 and 7 discuss pulmonary perfusion, regional differences thereof, the contribution of gravitational versus nongravitational influences, and the modulating influences of microgravity. The role of fractal principles is emphasized (a fractal structure is one that remains constant over a large range of scales). Chapters 8 and 9 discuss gas exchange at rest and during exercise, during normal, high, and low gravitation. These chapters present the data that support the concept that similar directional changes in overall ventilation and perfusion during microgravity result in only modest changes in gas exchange, and the concept that the lungs are optimally efficient at 1 G. Chapter 10 discusses central venous pressures and the pitfall of extrapolating super-G data to microgravity environments. This chapter includes an excellent discussion of the possible mechanisms underlying an apparent paradox observed during microgravity: central venous pressures are decreased in the setting of increased cardiac output. Chapter 11 details the complex interactive principles that determine pulmonary interstitial fluid fluxes and their modulation in microgravity. Chapter 12 discusses control of ventilation and reminds us of the important interaction between the cardiovascular and respiratory systems (baroreceptor reflex modulating influence on hypoxic respiratory drive). Chapter 13 discusses decompression sickness in the context of extra-vehicular activities, which involves transition from a cabin pressure of approximate atmospheric pressure to low atmospheric pressure (~30–40% of cabin pressure). Moreover, ground-based training includes long dives with changes in pressure from normal to high and back. This chapter provides an excellent and detailed description of decompression sickness and examines the apparent anomaly of almost no reports of decompression sickness in space, despite a sound predictive basis to the contrary.

The book concludes with an excellent summary chapter that deftly strikes a balance between concepts that are not yet completely reconciled, such as gravitational versus nongravitational contributions to pulmonary perfusion nonhomogeneity. This final chapter fittingly closes with an outline of seminal research questions that need to be addressed. Overall this book is a challenging read but well worth it. Though there is some modest and acknowledged overlap between certain chapters, this occurs only to the extent that it is likely to benefit (rather than irritate) the reader who has a broad interest in pulmonary physiology and disease.

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**Respiratory Physiology of Newborn Mammals: A Comparative Perspective.** Jacopo P Mortola MD. Baltimore, Maryland: Johns Hopkins University Press. 2001. Hard cover, illustrated, 344 pages, $89.95.

When I received **Respiratory Physiology of Newborn Mammals: A Comparative Perspective** my first thoughts were of my comparative anatomy course in college and quite frankly I was not looking forward to reading this book. But I must admit I found it very well written, informative, and enjoyable. As with many books, its creation began with lecture notes, in this case lecture notes on neonatal respiration. With expansion and additions the author has created a text that makes interesting comparative analysis of mammal species.

The book's primary objective was to discuss the main aspects of respiratory physiology of the developing mammal, including the human infant. The author achieved this by focusing principally on the mechanical, metabolic, and neural aspects of pulmonary ventilation. The book is written primarily for students in developmental physiology or comparative biology or zoology but also may be useful to neonatologists and pediatric pulmonologists keen on refreshing their appreciation of the basic concepts of their clinical practice.

The book includes a list of abbreviations and a glossary, which I found myself referring to frequently while reading some chapters. It would have been helpful to provide short definitions in the text and longer definitions in the glossary.

The book has a blue cover with silver lettering. The pages and print are of good quality and the binding is durable. The book is well referenced, with over 900 references. Borrowed tables, figures, and other material are used throughout the book, deriving from societies and organizations such as the American Pediatric Society, American Physiologic Society, American Thoracic Society, British Thoracic Society, Canadian National Research Council, National Geographic Society, Royal Society of Medicine, and the journal *Nature*.

The book is divided into 5 chapters: Gestation and Birth; Metabolic and Ventilatory Requirements; Mechanical Behavior of the Respiratory Pump; Reflex Control of the Breathing Pattern; and Changes in Temperature and Respiratory Gases. Chapter organization is consistent throughout the book. Each chapter concludes with 3 brief sections: Interspecies Comparisons; Clinical Implications; and Chapter Summary. I thought these sections were strong points in each chapter, providing the reader a quick, to-the-point version, with the chapter summary giving a final take-home-message. I found the text clearly written and concise, with no typographical or grammatical errors, and very well cross-referenced. There are a large number of tables and figures. It is obvious that great care was taken to ensure that the figures and tables were appropriate to the content of the chapters in which they appeared. They are very well done in that they are clear and readable, and the descriptions of the figures are well written.

There are a few photographs, and most are very clear. For example Figure 2.1 is a superb photograph of a dasyurid marsupial born after about 13 days gestation, in which the air sacs on each side of the heart are visible through the skin. On the other hand, Figure 4.19, which shows the time course of phrenic nerve development, has 3 small photographs of a rat embryo, and they are indistinguishable, although recognition of the details of the photographs is not essential to understand the essence of the figure.

The book opens with a comparison of various mammals' gestation and birth. Emphasis is on the development of the respiratory system, beginning with the respiratory system before birth and during birth, with the establishment of breathing control and lung volumes and capacities. The chapter ends with discussion of pulmonary circulation.

Chapter 2 looks at metabolic and ventilatory requirements, including such topics as pulmonary ventilation and mechanical events in the first hours after birth, pulmonary ventilation and the breathing pattern, dead space and alveolar ventilation, and coupling of ventilation and metabolism.

Chapter 3 is an extensive look at the mechanical behavior of the respiratory pump. After a brief description of respiratory muscle mass, fiber type, fatigue, and fatigue resistance, the chapter delves into subjects such as blood perfusion, the newborn thorax, mechanical interaction between lungs and chest wall, and static coupling between lungs and chest wall. There is further discussion of resistance to air flow, the active mechanical behavior, and energetics. I was disappointed in the brief presentation of topics such as resistance to air flow and time constant.

Chapter 4, "Reflex Control of the Breathing Pattern," covers such topics as reflex control of breathing amplitude and duration, ventilatory reflexes of extrapulmonary origin, central organization of respiratory neurons, and breathing during feeding.

Clinicians working in neonatal intensive care will find the last chapter especially interesting and clinically relevant to the development and care of the newborn infant. So much of the care of the newborn infant is directed toward maintaining cellular oxygenation and temperature, which is essential for survival. This chapter covers subjects such as change in ambient or body temperature, ventilatory control at various temperatures, oxygen and carbon dioxide chemoreceptors, changes in oxygenation, prenatal and acute neonatal hypoxia, chronic neonatal hypoxia, hyperoxia, and hypercarbia.

Following the last chapter there are 3 appendixes: "Passive Respiratory Mechanics: Some Applications to Measurements in Newborns," "Comparisons and Normalization," and "Orders and Suborders of the Class Mammalia." In the preface the author states, "Technical matters are usually not discussed, the chief exception being certain aspects of respiratory mechanics. The methodological details in Appendix A should facilitate an understanding of this topic." Appendix A covers compliance, resistance, time constant, and the relationship of time constant and resistance. I suspect that individuals who have little knowledge of this subject will find this section difficult to understand. To try to cover these complex topics in 8 pages is difficult. I found the information of little use for understanding the material in the preceding chapters.

Overall this book provides a wealth of information to identify and analyze the mechanisms that have evolved to guarantee adequate pulmonary ventilation in the neonatal mammal. This book should not be considered a standard respiratory text for a basic respiratory curriculum, but it would be valuable as a reference for clinicians working with newborn infants, as well as for those teaching neonatal respiratory medicine, to complement standard respiratory texts in bachelor of science and graduate level programs.

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**Exercise Physiology for Health Care Professionals.** Frank J Cerny PhD and Harold W Burton PhD. Champaign, Illinois: Human Kinetics. 2001. Hard cover, illustrated, 382 pages, $59.

**Exercise Physiology for Health Care Professionals** is a well-written elementary physiology text that covers the broad scope of physiology related to exercise. It is intended to provide the essential knowledge necessary for physical therapists, exercise scientists, and medical and dental students. The writing style is clear, concise, and consistent with the reading level of a beginning student. As the introduction indicates, the book provides "basic physiologic concepts as they apply to exercise, minimizing factual overload and maximizing application of the basic principles to exercise responses and adaptations under many conditions."

The book includes 5 chapters on energetics and metabolism, 2 on neural control and neuropathies, 3 on skeletal muscle and muscle diseases, 2 each on the pulmonary and cardiovascular systems and their diseases, 1 on pediatrics, 1 on aging, 1 on special environments, and 1 on exercise testing. Each chapter is 10–20 pages long. This broad brief coverage of physiology necessitates simplifications and didactic presentation of the concepts, which glosses over the complexity of the systems and leaves many questions. Simplicity is appropriate for a basic text whose stated goal is to present exercise physiology within a clinical context, "minimizing factual overload," but may leave the student with an overly simplistic comprehension and little appreciation for the complexity of an in-depth understanding. Nonetheless, a basic understanding of the fundamental concepts and the ability to apply them clinically may serve the beginning practitioner better than a more in-depth but less definitive explanation.

The most outstanding feature of this book is the inclusion of excellent clinical case studies with each concept. The case studies, incorporated within the text, are carefully designed to illustrate the principles mentioned above. Both the clinical presentation and its resolution are presented. For example, the discussion of the time course of glycolytic and oxidative metabolism in exercise includes 2 case studies. The first is of an athlete who consistently performs better in practice sessions than in competition. In resolving the case it is noted that the benefit of stimulating oxidative metabolism during the warm-up is lost if the warm-up is not immediately before the race—a situation frequently encountered in competition. The second case is a study of a marathon runner who fails to supplement his glucose intake throughout the run and thus exhausts his glycogen stores before completion of the race. In addition to the case studies within the text, each chapter includes case studies for the student to answer in review. Though when resolved the case studies are logical and illustrate the physiology just presented, I was uncertain that a student given the physiology as presented could independently reason through to the answer. The case studies do, however, provide students with solid practical examples of why they might want to know the physiology and how it is applicable to practice. Answers to the review questions are provided in an appendix. The case studies include decision-making with a broad spectrum of individuals, from ill-and-recovering to average to exceptionally athletic.

The book makes extensive use of education concepts. Each chapter begins with a bulleted list of 2 or 3 topics to be covered and concludes with a section entitled "What You Need to Know From Chapter X," which includes key terms and concepts the student should have learned. It does not provide the definitions nor a summary of the concepts but, instead, lists the terms and concepts for the student to review. Within the text the important concepts are briefly summarized in a smaller font, set off from the other text by lines. The book also makes extensive use of headings and subheadings to aid in organization. The figures are concise and uncluttered. A list of 10–20 references at the end of each chapter provides a starting place for further reading. References date from the 1970s through 2000, with the majority from the mid-1990s. They seem to represent both the use of classic material and the time lag between writing a book and its publication. The glossary provides ready access to definitions, and the index is complete and permits rapid access to particular subjects.

The book is a good overview of exercise physiology and brings the beginning student forward into the application of the physiologic concepts of exercise. This ability to translate scientific principles into clinical application is an enormous step, and its difficulty should not be underestimated. I would recommend this as an excellent book for the student who is beginning to study exercise physiology and for the professional who needs to understand the physiologic rationale for the prescription of exercise.

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**Textbook of Respiratory Medicine** CD-ROM, 3rd edition. John F Murray MD and Jay A Nadel MD, Editors. CD-ROM for Windows and Macintosh. Philadelphia: WB Saunders. 2001. $325.

This CD-ROM version of the 3rd edition of Murray and Nadel's **Textbook of Respiratory Medicine** is slightly more than an electronic reproduction of the 2-volume hard-cover textbook. As was the intention of the original text, this CD-ROM provides comprehensive and accurate information for medical care providers concerned with pulmonary diseases, and also serves as a valuable pulmonary medicine learning tool for house staff and medical students. Although there are detailed sections pertaining to pulmonary physiology and function, this is probably somewhat more detailed than necessary for respiratory technicians concerned with learning the basic physiology of pulmonary function. Included in the CD-ROM are over 400 tables and 900 images found in the original textbook. There are few features that make the CD-ROM version different from the paper edition.

The user's manual for the CD-ROM is straightforward and succinct, and the program is relatively self-explanatory. This CD-ROM contains versions for both Windows and Macintosh computers. The minimum Windows requirements are: Windows 95, 98, 2000, or NT; 200 MHz processor; 32 MB RAM; 30 MB hard drive space; and either Netscape 4.7 or Internet Explorer 5.0, with Java enabled. The minimum Macintosh requirements are: PowerPC; 200 MHz processor; Macintosh operating system 8.1; 30 MB RAM, 20 MB hard disk space; and Netscape 4.7, with Java enabled. The software loaded quickly and without difficulty on a Pentium III 850 MHz computer with Windows 2000, which I used for this review. Once loaded, the CD-ROM is always required for use of the program.

Upon starting the program the opening interface is a split screen—a popular format used by many electronic reference programs. Across the top of the right section there is a toolbar that contains some standard buttons such as "Main, Search, History, Bookmarks, Notes, Subject Index, Help, and Exit." The history function maintains a history of all activities in the program from the first time the program was initiated. The left side of the screen shows an outline of the book. When a part of the book is opened, subheadings are displayed. The main section of this opening page provides 4 options: browse respiratory medicine; search respiratory medicine; browse subject index; and using respiratory medicine. When selected, these either appear in the existing window or open another window. The first 3 selections are various options for accessing the medical content of the CD-ROM; the last selection is a help option for learning how to use the software.

The 3 search options are quite intuitive. The first and the last are identical to using either the content or index section of the textbook. The first method is browsing the content section, which is divided into parts, then sections, then chapters, then subheadings; this is a reproduction of the content outline in the textbook. To facilitate navigation the content is arranged in outline on the left side of the screen as the various parts are opened. This allows navigating through the text without having to go back to previous screens in the main section. Although searching by this method limits the reader to words contained in the heading of each section, this is slightly more efficient than the book, because one can view the subheadings of each chapter at a glance without having to read through the entire chapter.

The second method of finding information is the search function. Unfortunately, the search function in this program is rather elementary in that it does not recognize Boolean commands, thus making it inefficient for finding topics that are related to each other. In fact, it is quite similar to search functions found in standard Microsoft Word and Adobe Acrobat, although it will recognize standard medical terms.

The third method is using the index, as one traditionally does when using the textbook. Here there are no surprises. The index is identical to that found in the textbook. A helpful addition again is the outline to the side of the main screen. Once an item is selected from the index, the software immediately locates the item in the outline to the left and displays the entire text section in the main screen.

Once the user has entered the text, the layout of each page is mostly identical to the textbook. One difference is the display of tables and figures, which appear as thumbnails embedded in the text, with either a highlighted link (tables) or a box with the associated text to the right (figures). Clicking the link or figure opens a new window with a full reproduction of the table or figure. The contents of the tables are identical to that found in the text, although the coloring of the tables is slightly different. The figures are crisp, clear reproductions of those found in the text, including the radiographs. There are no color enhancements to the figures. As in the textbook, the pathology figures are in black-and-white.

The program maintains a list of bookmarks that one can insert on any page of the text, but unfortunately these bookmarks can not be inserted at specific lines or words in the text. The bookmark function is complemented by the notes function. The reader can create a note for the active page in the notes box at the bottom of the page. All notes created since the program was first used can be displayed, in alphabetical order, by clicking on the notes button in the toolbar. There is no option to display these notes by subject or date created. Another option with this software is the ability to copy and paste any portion of the text, including tables and figures, into another file. However, one should be cautious to respect the copyright laws. Finally, there are over 10,000 references within this program, and they are linked to their primary MEDLINE abstracts, allowing easy access to the original references for more in-depth review.

In summary, the CD-ROM version of the 3rd edition of Murray and Nadel's **Textbook of Respiratory Medicine** is an accurate electronic reproduction of the textbook. The program is different from the textbook in only a few features: direct link of references to MEDLINE abstracts, the ability to search key words, the ability to display the outline of the book while browsing the text, and the option to electronically copy portions of the book into other files. I did not consider the bookmark and notes functions a unique feature, because most readers of textbooks use bookmarks and write in the margins. Although the program meets its primary objectives, these are rather basic. It would have been more justifiable to create an electronic version of the textbook if the creators were able to take full advantage of the technology available as a result of the digital revolution. Respiratory medicine is a dynamic field that can benefit from the standard technology now found in most electronic publications, such as animations of physiologic functions, color-enhanced anatomy figures, and color reproductions of pathology specimens. The question of whether this CD-ROM is worthy of purchase should be answered according to the individual's needs and preferences. Certainly the CD-ROM is not so unique that one needs to own both the CD-ROM and the printed version, especially when each sells for over $300. The electronic version is portable and efficient, ideal for a student or physician who uses a laptop computer. However, for the more traditional user, the few electronic features unique to this version do not outweigh the satisfaction of holding such an immense body of knowledge in one's hands, and actually being able to experience the sensation of flipping a page.

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**Atlas of Procedures in Respiratory Medicine: A Companion to Murray and Nadel's Textbook of Respiratory Medicine.** Warren M Gold MD, John F Murray MD, and Jay A Nadel MD. Philadelphia: WB Saunders. 2002. Hard cover, illustrated, 508 pages, $139.

The **Atlas of Procedures in Respiratory Medicine** by Gold, Murray, and Nadel is a welcome addition to the outstanding *Textbook of Respiratory Medicine* by Murray and Nadel. The authors' intent was to provide a companion "picture book that illustrates how things are done in respiratory medicine." I think they have done a good job.

The atlas is divided into 8 chapters and 20 appendixes. The chapters cover: morphological procedures, anatomy, pathology, radiography, nuclear medicine, bronchoscopy, microbiology, and pulmonary function testing (PFT). The appendixes list reference equations and predicted normal values. Conspicuously absent are chapters on pleural procedures and ventilators. The references throughout the book are well selected and numerous. The index is extensive and very usable.

The pictures are of very high quality and are a goldmine, especially for pulmonary fellows. I recall my mentor in pulmonary fellowship telling me that a great way to get a "leg up" for the boards was to collect all the pictures of the various infectious diseases and malignancies we encounter as pulmonologists. The questions frequently involved a very long stem associated with a picture of an organism or tumor. One could glean the answer from the stem, but if you could recognize the "bug" or tumor in the picture, you'd get the answer much more quickly. I dutifully collected these pictures from various sources (infectious disease textbooks, pathology atlases, monographs, etc) and found out that he was correct. Recognizing the pictures helped a lot. Well, now pulmonary fellows have the pictures collected without having to do the collecting!

The tables are constructed very logically, with enough text to make them easily navigable. I was surprised that the authors left out the recently updated lung cancer staging system in both the pathology and radiology sections.[1] On the other hand, there are some wonderful tables, including some that contrast the various clinical and pathology features of the noninfectious interstitial pneumonias. The first chapter, on morphological procedures in respiratory anatomy and pathology, would be quite helpful for a research fellow looking for a technique to evaluate the lung in a protocol. For the clinician, the chapter on microbiology laboratory diagnosis is an outstanding resource on how to collect and evaluate respiratory specimens for various infectious diseases.

The radiography section is very up to date in that it includes a section on digital radiography, which is rapidly entering our daily practice. There are some nice examples of the clinical utility of high-resolution computed tomography, although I would have liked some more (such as of emphysema or cystic disease). The chapter on nuclear medicine has one of the best collections I have seen of abnormal ventilation/perfusion scans. The correlation with anatomy is excellent. This section may become rapidly outdated, with newer high-speed computed tomography scanners doing pulmonary angiograms, which did not get near as much coverage in this atlas. There is also a nice section on positron emission tomography scanning, which is being used more and more in clinical practice.

The chapter on bronchoscopy, written by Udaya Prakash and Sergio Cavaliere, is wonderful, with many very helpful endobronchial anatomy pictures, and it includes a unique section on endobronchial ultrasound. There is an extensive section on therapeutic bronchoscopy, which would be extremely helpful to the novice pulmonary fellow or the practicing clinical pulmonologist learning new techniques. The sections on collection, preservation, and transport of respiratory tract specimens would be very helpful to bronchoscopy technicians, to make sure the specimens are handled properly.

The last chapter is on PFT and was written by the lead editor, Warren Gold. His tables describing pressure and volume plethysmographs are the most readable and understandable that I have come across. I showed them to our registered pulmonary function technicians and they agreed with me. The chapter is very complete and covers just about every pulmonary function test that I could think of, and some more. The chapter has many tables, with a lot of very practical advice on how to perform the tests as well as interpret them. The section on exercise with clinical applications is easy to read and readily usable. The chapter is clearly evidence-based, and areas of controversy such as the anaerobic threshold are treated fairly. The one problem I have with the PFT chapter is that the section on challenge testing did not include the American Thoracic Society's recent guidelines for methacholine and exercise challenge testing.[2] I had hoped that those guidelines would bring some order to this procedure. Dr Gold recommends using body plethysmography and airway resistance for challenge testing, whereas the guideline recommends using airway resistance only as an alternative to spirometry with patients unable to perform spirometry. The beauty and simplicity of most of his other explanations make up for this difference of opinion.

In conclusion, this atlas would be a welcome addition to any pulmonologist's library and probably should be considered a must-have for those taking the pulmonary certification exam. Practicing respiratory therapists may not find much useful information, but pulmonary function technicians and bronchoscopy technicians will probably find a lot of useful information in this very attractive picture atlas.

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*Note: The views expressed herein reflect only the views of the author and are not the official views of the Department of the Army or the Department of Defense.*

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**Respiratory Nursing**. Glenda Esmond BSc DiP Nursing RGN, editor. Edinburgh, United Kingdom: Ballière Tindall. 2001. Hard cover, illustrated, 271 pages, $40.

**Respiratory Nursing** was written primarily for nurses, with content covering the basics of respiratory science and nursing care. However, the content may also be applicable for the respiratory therapist, who is often involved in the bedside care of patients with respiratory problems.

The content includes a comprehensive chapter on respiratory anatomy and physiology, followed by chapters on assessment, diagnostics, respiratory support techniques, respiratory infections, and oxygen therapy. What renders this book specific to nursing is its emphasis on the psychological and social ramifications of chronic lung disease. Chapters dedicated to this include a well-written, comprehensive, and useful section on smoking and smoking cessation, as well as chapters on living with chronic respiratory illness, nutrition, pulmonary rehabilitation, and end-stage management of respiratory disease. These chapters are patient and family focused and are geared toward empowering health professionals to help patients effectively manage respiratory illness, through patient and family education and knowledgeable, compassionate nursing care.

The editor, Glenda Esmond, prefaces the text with an overview of respiratory illnesses in the United Kingdom and the ramifications to that society. The statistics cited are not absolutely applicable to lung disease in the United States, but the reader can generalize the findings to the United States population and can appreciate when Esmond states, "It is likely that the psychological and economic costs as a result of respiratory illness will continue, and indeed will most likely escalate in the future."

The material is well selected and organized. Each chapter starts with a boxed section that outlines the chapter contents. Throughout the text are boxes outlining major teaching points within the text. The illustrations are clear and easily understandable. There is an excellent patient teaching section on inhaler and nebulizer devices. Throughout the text are case studies that highlight information from the text. The references are generally recent, but some date back to the early 1990s or even 1980s. Additional information on and examples of chest computed tomography would be helpful, as this diagnostic tool is becoming more available and common. A section on caring for the patient after thoracic surgery would also be useful, and especially lacking is a section on chest tube management.

Because the text is geared toward the United Kingdom practitioner, some details may be confusing or distracting to the United States reader. Certain drugs mentioned, such as oxitropium, are not available or only very recently available in the United States and will be unfamiliar to many practitioners. Some units of measure, such as kPa (used for blood gas values), are not routinely used in the United States. Oxygen delivery systems are different in the United States, as are many of the featured noninvasive ventilators. Chapter 13, "Primary and Secondary Care Interface," which deals with United Kingdom health policy and public health framework, is largely irrelevant to the United States reader. What is missing and important for the United States reader is information regarding Medicare and Medicaid reimbursement policies, especially for home oxygen and noninvasive ventilatory support systems. Another thing that may confuse United States practitioners is the British Thoracic Society guidelines for asthma, as United States practitioners rely on the 1997 National Institutes of Health guidelines. Practitioners in the United States also rely on treatment guidelines for community-acquired pneumonia that stratify disease acuteness into specific treatment decision trees. This is not referred to in the chapter on respiratory infections.

In general, the book is an enjoyable read. The paperback edition is very portable and therefore can be easily used as a reference book. As mentioned previously, the book is specific to nursing because of its emphasis on the psychosocial aspects of living with chronic respiratory disease, as well as patient and family teaching.

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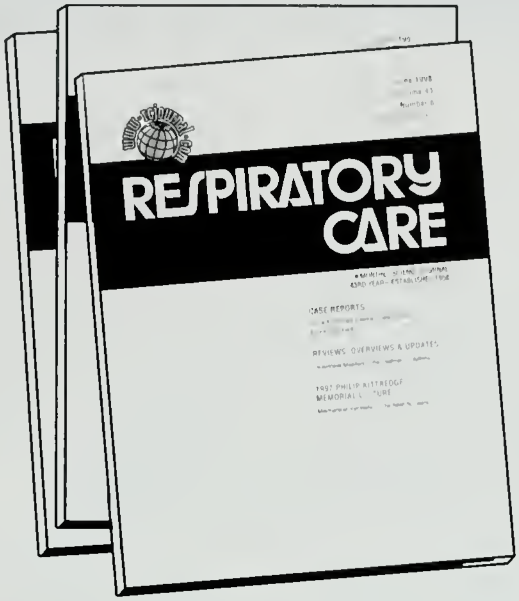
University of Virginia Health System
Charlottesville, Virginia



## *CRCE through the Journal—2002*

### Answer Key

For your information, the correct answers to the 50 question for *CRCE through the Journal*, which appeared in the August 2002 issue of RESPIRATORY CARE, are given below. Deadline for submission of answer sheets for CRCE credit was September 30, 2002.

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| 1. | B | 11. | A | 21. | A | 31. | C | 41. | C |
| 2. | D | 12. | A | 22. | D | 32. | D | 42. | A |
| 3. | D | 13. | B | 23. | C | 33. | D | 43. | C |
| 4. | B | 14. | C | 24. | B | 34. | A | 44. | A |
| 5. | C | 15. | A | 25. | B | 35. | D | 45. | B |
| 6. | C | 16. | D | 26. | A | 36. | B | 46. | D |
| 7. | A | 17. | C | 27. | B | 37. | A | 47. | C |
| 8. | D | 18. | A | 28. | C | 38. | D | 48. | A |
| 9. | B | 19. | C | 29. | B | 39. | C | 49. | D |
| 10. | A | 20. | D | 30. | B | 40. | C | 50. | B |

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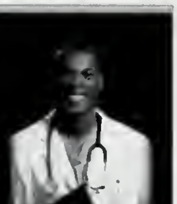

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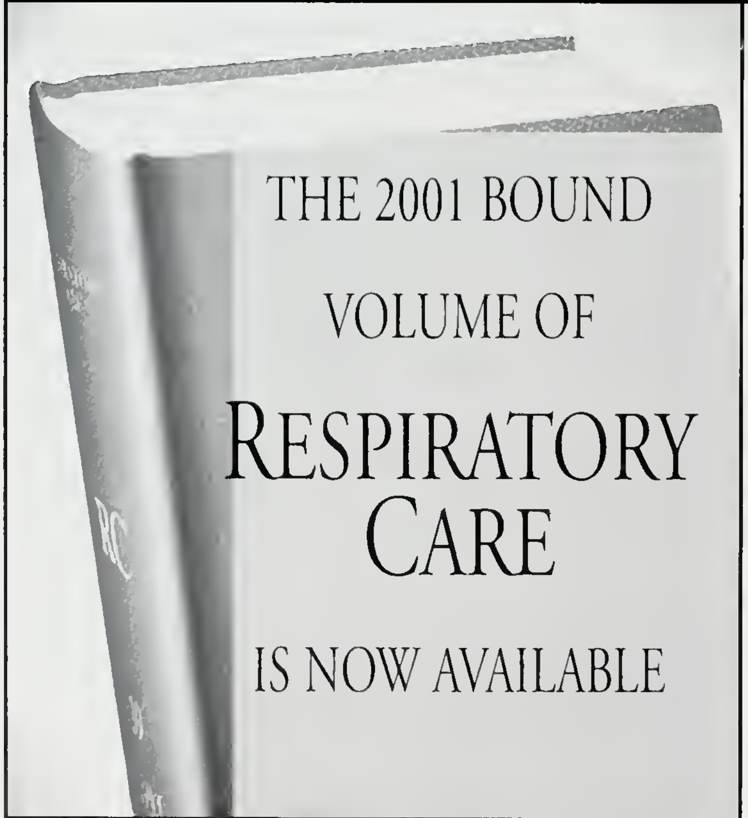

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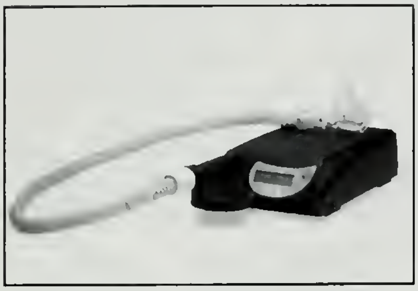
News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE, New Products & Services Dept, 11030 Ables Lane, Dallas TX 75229-4593. The Reader Service Card can be found at the back of the Journal

# New Products & Services



**At-Home Ventilation.** Weinmann GmbH+Co, announces a new device for bi-level at-home ventilation, the Somnovent® ST. Thanks to its sensitive trigger, claims the manufacturer, the Somnovent ST adjusts to each patient's individual respiratory characteristics and runs at a pleasantly low sound level. Weinmann says that in addition to its use in treating sleep-related respiratory disruptions, Somnovent ST can be used by patients with general respiratory insufficiency, such as COPD and OSAS; respiratory mechanism (scoliosis) and neuromuscular disorders; and by patients with partial central respiratory disorders. For more information from Weinmann GmbH+Co, circle number 188 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

**Tracheostomy Straps.** DHD Healthcare Corp's tracheostomy strap features soft foam padding and convenient, adjustable Velcro hooks. According to DHD, it fits all patients and has no sharp edges or twill ties; thus, it is ideal for long-term patient care. DHD recently acquired the strap from STI Medical Products Corp. For more information from DHD Healthcare Corp, circle number 189 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

**Full Face Mask.** The 7600 Series Vmask™ from Hans Rudolph is a full face mask for use with CPAP/NPPV blowers and ventilators by OSA patients and is available in 5 sizes. According to Hans Rudolph, the face piece and sealign flange are integrally molded of soft, clear, silicone rubber in an anatomically contoured design with ribbed support for a leak-free comfortable fit. The swivel port elbow assembly features a safety breathe-out valve, and the mask face piece has $CO_2$ flush holes for safety. The quick-release headgear was designed for patient comfort and safety and is available in 2 sizes, says Hans Rudolph. For more information from Hans Rudolph, circle number 190 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

**Dual Parameter Monitor.** Nonin Medical Inc has introduced their noninvasive blood pressure monitor and digital pulse oximeter. According to the company, the Avant™ 2120 combines Nonin's proven pulse oximetry with fast, motion-tolerant oscillometrip NIBP that results in a compact, lightweight, cost-effective, and reliable health care tool. The manufacturer says that the device can be used on pediatric patients as well as adults. Accessories include a broad range of blood pressure cuffs, various oximetry sensors, rolling stands, and a carrying case. For more information from Nonin Medical, circle number 191 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

# Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

## *Scheduled Professor's Rounds 2002*

**Withholding and Withdrawing Life Support in the ICU** — Gordon D Rubenfeld MDMSc / Richard D Branson BA RRT FAARC — Videotape Available

**Weaning from Mechanical Ventilation: New Insights, New Guidelines** — Neil R MacIntyre MD FAARC/ Dean R Hess PhD RRT FAARC — Videotape Available

**Neonatal and Pediatric Ventilators: What's the Difference?** — Mark J Heulitt MD FAARC/ Richard D Branson BA RRT FAARC — Videotape Available

**Ventilator Graphs: What's With That Wave?** — Jon O Nilsestuen PhD RRT FAARC/ Richard D Branson BA RRT FAARC — Videotape Available

**Talking with Patients and Families About Death and Dying**— Helen M Sorensen MA RRT FAARC/ David J Pierson MD FAARC — Videotape Available

**Pressure vs Volume Ventilation: Does It Matter?** — Robert S Campbell RRT FAARC/ Richard D Branson BA RRT FAARC — Live September 10/ Audio October 8

**Inpatient Management of COPD** — Randall Rosenblatt MD/ David J Pierson MD FAARC — Live October 22; Audio November 12

**High-Frequency Oscillatory Ventilation** — Thomas E Stewart MD/ Richard D Branson BA RRT FAARC — Live November 19; Audio December 10

**United States Postal Service**
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| **a** Total No of Copies (Net Press Run) | 28,632 | 27,548 |
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| **d.** Free Distribution by Mail | 617 | 636 |
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| **g** Total Distribution | 28,236 | 27,203 |
| **h.** Copies Not Distributed | | |
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**17** Signature and Title of Editor, Publisher, Business Manager or Owner
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I certify that all information furnished on this form is true and complete

Ray Masferrer

### The National Board for Respiratory Care— Examination Fees for 2002

| Examination | Examination Fees |
|---|---|
| CRT | $190 (new applicant)<br>$150 (reapplicant) |
| Perinatal/Pediatric | $250 (new applicant)<br>$220 (reapplicant) |
| CPFT | $200 (new applicant)<br>$170 (reapplicant) |
| RPFT | $250 (new applicant)<br>$220 (reapplicant) |
| RRT (Written & CSE) | $190 (new) $150 (reapplicant) written only<br>$200 (new and reapplicant) CSE only<br>$390 (new) $350 (reapplicant) both |

For information about other services or fees, write to the National Board for Respiratory Care,
8310 Nieman Road, Lenexa KS 66214, or call
(913) 599-4200, FAX (913) 541-0156,
or e-mail: nbrc-info@nbrc.org

American Association for Respiratory Care

# MEMBERSHIP APPLICATION

### ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC

### ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field

### STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member

Please read the eligibility requirements for each of the classifications above, then complete the form. All information requested must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

☐ Active
Associate
    ☐ Foreign
    ☐ Physician
    ☐ Industrial
☐ Special
☐ Student

Last Name ______________________________

First Name ______________________________

Social Security No. ______________________________

Home Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __ - __ __ __ __

Phone No. ( ______ ) ______________________________

You are automatically assigned to a state society based on your home address. If you wish to be assigned to a different state society, please indicate which state that is here:______________

**Work Information:**

Place of Employment ______________________________

Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __ - __ __ __ __

Phone No. ( ______ ) ______________________________

Preferred Fax No.( ______ ) ______________________________

Preferred Email Address ______________________________

Preferred mailing address: ☐ Home ☐ Business

Would you like to receive our monthly newsletter, AARC Report, by email?
☐ Yes ☐ No

Have you ever been or are you currently in the military?
☐ Yes ☐ No

## Demographic Questions (optional)

We request that you answer these questions in order to help us design services and programs to meet your needs.

**Primary Job Responsibility (check one only)**

☐ Director (Technical or Program)
☐ Supervisor
☐ Diagnostic Technologist
☐ Instructor/Educator
☐ Other, specify ______________
☐ Therapist/Technician
☐ Medical Director
☐ Student
☐ Staff Nurse

**Type of Business**

☐ Educational Institution
☐ DME/HME
☐ Home Health Agency
☐ Hospital/Acute Care
☐ Other, specify ______________
☐ Manufacturer or supplier
☐ Outpatient Clinic
☐ Physician office
☐ Skilled Nursing Facility

**Check the Highest Degree Earned**

☐ High School
☐ RC Graduate Technician
☐ Associate Degree
☐ Bachelor's Degree
☐ Master's Degree
☐ Doctorate Degree

**Number of Years in Respiratory Care**

☐ 0-2 years
☐ 3-5 years
☐ 6-10 years
☐ 11-15 Years
☐ 16 years or more

**Job Status**

☐ Full Time ☐ Part Time

**Credentials**

☐ RRT
☐ CRT
☐ Physician
☐ CRNA
☐ RN
☐ LVN/LPN
☐ CPFT
☐ RPFT
☐ Perinatal/Pediatric

Date of Birth ____________ Sex __________

***FOR STUDENT MEMBER – REQUIRED***

School/RC Program ______________________________

Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __ - __ __ __ __

Phone No. ( ______ ) ______________________________

**Expected Date of Graduation** (REQUIRED INFORMATION)

Month _______ Year _______

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# merican Association for Respiratory Care **MEMBERSHIP APPLICATION**

## embership Fees

yment must accompany your application to the AARC. Fees are r 12 months. These fees contain the $12.50 new members ocessing fee. Renewing members (except students) can deduct 12.50.

CHOOSE ONE LEVEL OF MEMBERSHIP

ARC REGULAR MEMBERSHIP (Receive both AARC Times and spiratory Care journal)

| | |
|---|---|
| ☐ Active | $102.50 |
| ☐ Associate (Industrial or Physician) | $102.50 |
| ☐ Associate (Foreign) | $117.50 |
| ☐ Special | $102.50 |
| ☐ Student | $ 50.00 |

**OR**

ARC CHOICE MEMBERSHIP(Choose one publication)

| | |
|---|---|
| ☐ Active | $ 91.00 |
| ☐ Associate (Industrial or Physician) | $ 91.00 |
| ☐ Associate (Foreign) | $106.00 |
| ☐ Special | $ 91.00 |

want ☐ AARC Times ☐ Respiratory Care journal

**OR**

ARC PLUS MEMBERSHIP (All publications and other items)

| | |
|---|---|
| ☐ Active | $137.50 |
| ☐ Associate (Industrial or Physician) | $137.50 |
| ☐ Associate (Foreign) | $177.50 |
| ☐ Special | $137.50 |

(Includes one free section - please mark choice below.)

## *pecialty Sections (optional)*

stablished to recognize the specialty areas of respiratory care, ese sections publish a newsletter four times a year that focuses on sues of specific concern to that specialty. The sections also design e specialty programming at the national AARC meetings.

| | |
|---|---|
| ☐ Adult Acute Care Section | $15.00 |
| ☐ Education Section | $20.00 |
| ☐ Perinatal-Pediatric Section | $15.00 |
| ☐ Diagnostics Section | $15.00 |
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| ☐ Transport Section | $15.00 |
| ☐ Home Care Section | $15.00 |
| ☐ Subacute Care Section | $15.00 |

### PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to Respiratory Care journal and AARC Times magazine includes an allocation of $11.50 from my dues for each of these publications

*NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%*

**Signature** ____________________

**Date** ____________________

TOTAL MEMBERSHIP FEE $__________

TOTAL SECTION FEE $__________

**GRAND TOTAL = Membership Fee plus optional sections**

$__________

☐ Total Amount Enclosed $__________

☐ Please charge my dues (see below)

To charge your dues, complete the following:

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☐ Visa

Card Number

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Card Expires ______/______

Signature ____________________

**Mail application and appropriate fees to:**

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RESPIRATORY CARE

# Manuscript Preparation Guide

RESPIRATORY CARE welcomes original manuscripts related to the science and technology of respiratory care and prepared according to the following instructions and the *Uniform Requirements for Manuscripts Submitted to Biomedical Journals* (available at *http://www.icmje.org/index.html*). Manuscripts are blinded and reviewed by professionals with experience in the subject of the paper. Authors are responsible for obtaining written permission from the original copyright holder to use previously published figures and tables. Before publication, authors receive page proofs and are allowed to make only minor corrections. Accepted manuscripts are copy-edited for clarity, concision, and consistency with RESPIRATORY CARE's format. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission. Editorial consultation is available at any stage of planning or writing: contact the Editorial Office, 600 Ninth Avenue, Suite 702, Seattle WA 98104, (206) 223-0558, fax (206) 223-0563, E-mail: rcjournal@aarc.org

### Categories of Articles

**Research Article:** A report of an original investigation (a study). Must include: Title Page, Abstract, Key Words, Introduction, Methods, Results, Discussion, Conclusions, and References. May also include: Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendixes.

**Review:** A comprehensive, critical review of the literature and state-of-the-art summary of a topic that has been the subject of at least 40 published research articles. Must include: Title Page, Outline, Abstract, Key Words, Introduction, Review of the Literature, Summary, and References. May also include: Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendixes.

**Overview:** A critical review of a pertinent topic that has fewer than 40 published research articles. Same structure as Review Article.

**Update:** A report of subsequent developments in a topic that has been critically reviewed in RESPIRATORY CARE or elsewhere. Same structure as a Review Article.

**Special Article:** A pertinent paper not fitting one of the other categories. Consult with the Editor before writing or submitting such a paper.

**Editorial:** A paper addressing an issue in the practice or administration of respiratory care. It may present an opposing opinion, clarify a position, or bring a problem into focus.

**Letter:** A brief, signed communication responding to an item published in RESPIRATORY CARE or about other pertinent topics. Tables, Figures, and References may be included. The letter should be marked "For Publication."

**Case Report:** Report of an uncommon clinical case or a new or improved method of management or treatment. A case-managing physician must either be an author or furnish a letter approving the manuscript. Must include: Title Page, Abstract, Key Words, Introduction, Case Summary, Discussion, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

**Point-of-View:** A paper expressing personal but substantiated opinions on a pertinent topic. Must include: Title Page, Text, and References. May also include: Tables and Figures (if so, must include Figure Legends).

**Drug Capsule:** A miniature review paper about a drug or class of drugs. Drug Capsules address pharmacology, pharmacokinetics, and/or pharmacotherapy.

**Graphics Corner:** A brief, instructive case report discussing and illustrating waveforms for monitoring or diagnosis. Must include: Questions, Answers, and Discussion.

**PFT Corner:** A brief, instructive case report arising from pulmonary function testing, accompanied by a review of the relevant physiology and appropriate references to the literature. Must include: Questions, Answers, and Discussion.

**Test Your Radiologic Skill:** A brief, instructive case report pertinent to respiratory care and involving imaging, including one or more radiographs or other images submitted as black and white glossy photographs that clearly illustrate the teaching points being made. Must include: Questions, Answers, and Discussion.

### Preparing the Manuscript

Double-space the text and number the pages. Do *not* include author names, author institutional affiliations, or allusions to institutional affiliations anywhere except on the title page. On the Abstract page include the title but do not include author names. Begin each of the following on a new page: Title Page, Abstract, Text, Acknowledgments, References, each Table, each Figure, and each Appendix. Use standard English in the first person and active voice. Type all headings in initial-capital letters (eg, Introduction, Methods, Patients, Equipment, Statistical Analysis, Results, Discussion). Center the main section headings and place second-level headings on the left margin.

**Abstract**. Please ensure that the abstract does not contain any facts or conclusions that do not also appear in the body text. Limit the abstract to no more than 250 words.

**Key Words**. Include a list of 6 to 10 key words or key phrases in Research Articles, Reviews, Overviews, Special Articles, and Case Reports. Key words are best selected from the Medical Subject Headings (MeSH) used by MEDLINE and available at *http://www.nlm.nih.gov/mesh/meshhome.html*.

**References.** Assign reference numbers in the order that articles are cited in your manuscript. At the end of the manuscript, list the cited works in numerical order. Abbreviate journal names as in *Index Medicus*. List all authors. If the research has not yet been accepted for publication, cite the research as a personal communication (eg, Smith KR, personal communication, 2001); *however, you must obtain written permission from the author to cite his or her unpublished data.* Do not number such references; instead, make parenthetical reference in the body text of your manuscript. Example: "Recently, Jones et al found this treatment effective in 45 of 83 patients (Jones HI, personal communication, 2000)."

**The following examples show RESPIRATORY CARE's style for references.**

Paper accepted but not yet published:
Hess D. New therapies for asthma. Respir Care (year, in press).

Article in a journal carrying pagination throughout the volume:
Legere BM, Kavuru MS. Pulmonary function in obesity. Respir Care 2000;45(8):967-968.

Article in a publication that numbers each issue beginning with Page 1:
Kallstrom TJ. Focus on asthma—disease management: a role for the respiratory therapist. AARC Times 1999;23(Oct):16, 17, 19.

Corporate author journal article:
American Association for Respiratory Care. Clinical Practice Guideline. Removal of the endotracheal tube. Respir Care 1999;44(1):85-90.

Article in journal supplement: (Journals differ in numbering and identifying supplements. Supply information sufficient to allow retrieval.)
Barnes PJ. Endogenous inhibitory mechanisms in asthma. Am J Respir Crit Care Med 2000; 161(3 Pt 2):S176-S181.

Abstract in journal: (Abstracts citations are to be avoided, and those more than 3 years old should not be cited.)
Volsko TA, De Fiore J, Chatburn RL. Acapella vs flutter: performance comparison (abstract). Respir Care 2000;45(8):991.

Editorial in a journal:
Giordano SP. What's that sound? (editorial) Respir Care 2000;45(10):1167-1168.

Editorial with no author given:
The perils of paediatric research (editorial). Lancet 1999;353(9154):685.

Letter in journal:
Piper SD. Testing conditions for nebulizers (letter). Respir Care 2000;45(8):971.

Book: (For any book, specific pages should be cited whenever reference is made to specific statements or other content.)
Cairo JM, Pilbeam SP. Mosby's respiratory care equipment, 6th ed. St Louis: Mosby; 1999:76-85.

Corporate author book:
American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Chapter in book with editor(s):
Isono S. Upper airway muscle function during sleep. In: Loughlin GM, Carroll JL, Marcus CL, editors. Sleep and breathing in children: a developmental approach. (Lung Biology in Health and Disease, Vol 147, Claude Lenfant, Executive Editor.) New York/Basel: Marcel Dekker; 2000:261-291.

World Wide Web
American Lung Association. Trends in pneumonia, influenza, and acute respiratory conditions mortality and morbidity. February, 2000. http://www.lungusa.org/data. Accessed November 20, 2000.

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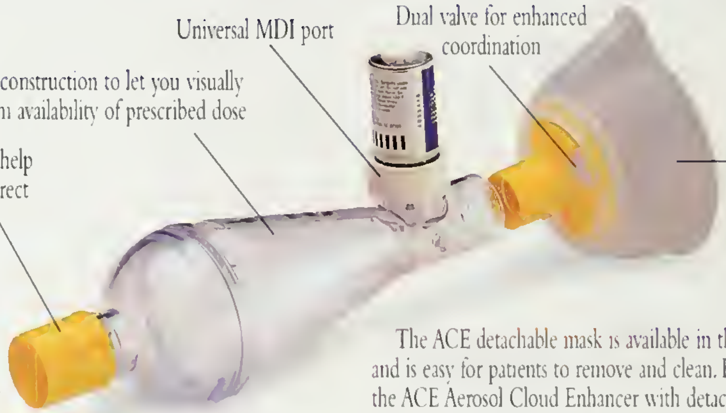
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